

# PRINCIPLES OF PLANT PATHOLOGY

BY

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## PREFACE

In planning this book the author has had in mind the building up of a general one-term or one-semester course in plant pathology to meet the needs of undergraduates in schools of agriculture. At the same time it is believed that the material included in this text can be used with as much success in non-technical colleges and universities where applied courses in botany are offered, as in agricultural schools. The writer has taught plant pathology and botany in an agricultural college for fifteen years. Whatever of merit this volume may contain is derived from that experience.

The author believes that a course of this kind should be more than a mere catalog of plant diseases with control measures appended. Graduates in agriculture should be expected to know *why* as well as *what* and *how* with respect to measures employed in the control of plant diseases. For this reason an attempt has been made to stress the principles underlying the cause and control of diseases in plants. However, in order that these general laws may be more thoroughly understood, it is necessary to have a knowledge of a considerable number of details concerning specific diseases. As a basis for making intelligent generalizations, a number of representative plant diseases have been outlined for study. Accordingly, the treatment of the subject naturally falls into two parts: (a) that treating of the general aspects of plant pathology; and (b) that dealing with the specific diseases selected for detailed study.

*Part I.* — The first part of the book consists of a discussion of topics of general interest concerning the nature, cause, and control of plant diseases. The subject matter of Part I includes such topics as: historical account of the rise and development of the science of plant pathology; definition, symptoms, and classification of plant diseases; relation of plant diseases to the environment; methods of investigating plant diseases; sick soil conditions; relation of insects to plant diseases; storage, transportation, and marketing problems arising from plant diseases; and control measures, such as the use of fungicides, disease-resistant plants, clean seed and nursery stock, and quarantine and inspection. Some of the chapters in Part I can be read to advantage early in the term; while others, perhaps, can be more fully comprehended if read later in the term after a considerable amount of laboratory work has been done so as to furnish a background of specific knowledge of particular diseases.

*Part II.* — The diseases selected for study in Part II have been chosen from as wide a field as possible, both as to types of causal agents and of symptoms represented, and as to range of host plants. The number of diseases that can be studied in such a course is quite limited. It has been the writer's aim to include a somewhat larger list of diseases than most classes will cover in a term's work so that some choice can be made. At the end of each chapter a supplementary list of diseases with references has been included for the benefit of those who desire a still more comprehensive range of material.

The diseases discussed in this part of the book are classified on the basis of *cause*. In the beginning course, particularly, it is believed that the *cause* of diseases should be emphasized, not from the mycological viewpoint, but simply as a matter of understanding the different types of causal agents as a basis for the intelligent application of control measures. The methods of control which are suitable or effective in any particular case are not dependent upon the symptoms of the disease but upon the nature and life history of the causal organism.

Under each disease listed in Part II, the treatment falls into four subdivisions: (a) a text discussion of the disease; (b) directions for laboratory study; (c) a list of review questions; and (d) a list of references.

Directions for laboratory work have been included for the benefit of those teachers who have not the time or do not care to make their own laboratory outlines. The outlines have purposely been made brief in order that they may be included without making the book unwieldy in size. It is felt that, with the text material at hand for ready reference, lengthy and detailed laboratory directions are not necessary. For those teachers who prefer to use their own laboratory outlines, these exercises may be ignored and will in no way interfere with the use of the book as a text.

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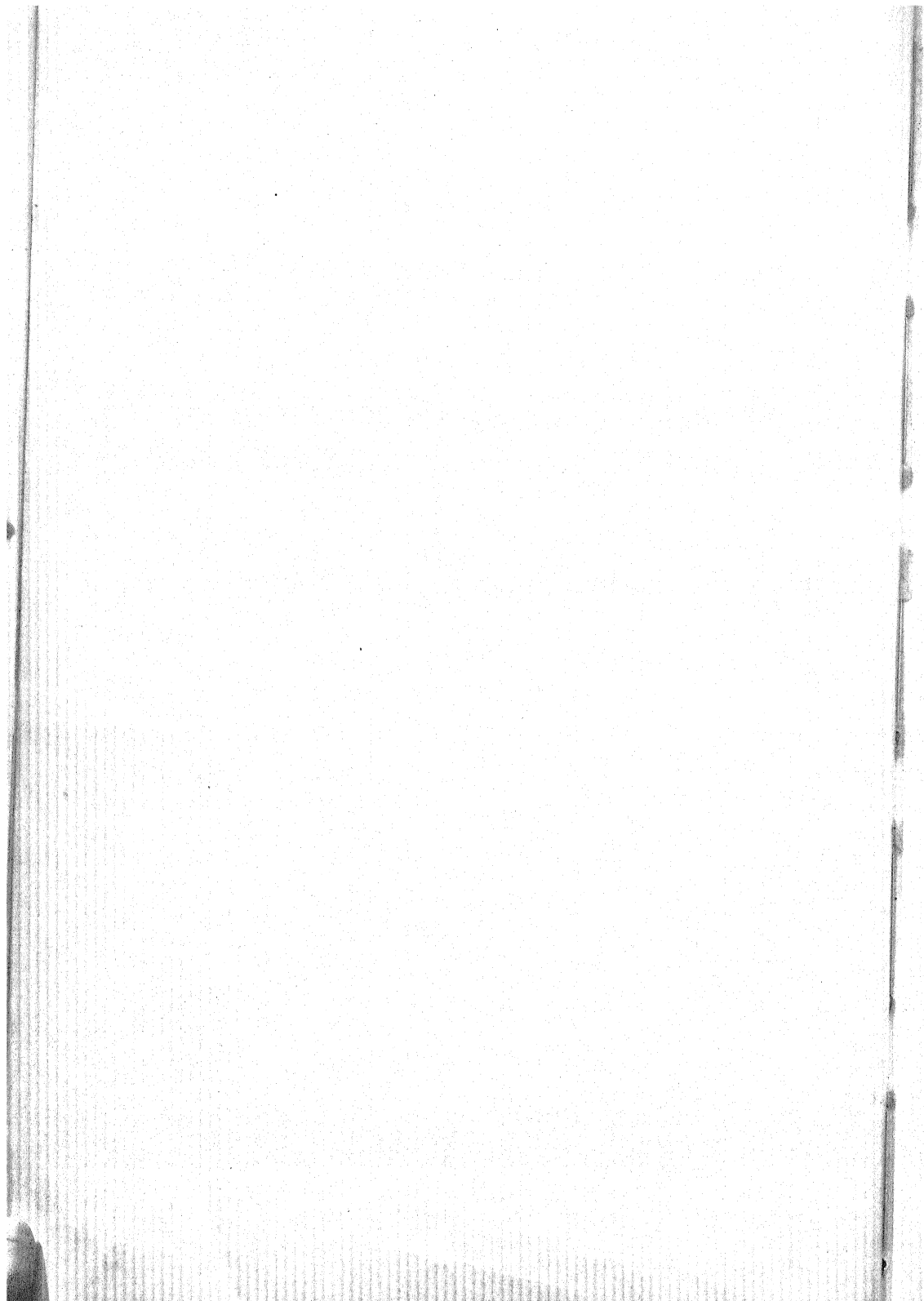
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# PRINCIPLES OF PLANT PATHOLOGY

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## PART I

### CHAPTER I

#### INTRODUCTION: RISE AND DEVELOPMENT OF PLANT PATHOLOGY

Plant diseases have been known since the dawn of history. References to blights, mildews and rusts occur in the Bible and in writings of the ancient Greeks and Romans. These early records show that, while the ancient peoples recognized that plant diseases were taking toll of their crops, they had no true conception of the nature and cause of these maladies, and their ideas concerning them were largely of a superstitious nature. Throughout the ages down to the middle of the nineteenth century the origin of disease in plants was shrouded in mystery. Many groundless theories were advanced to explain the occurrence of the observed phenomena. The general tendency was to attribute the troubles to God or the gods, as the case might be, or to the influence of the stars and planets. In the groping of these early agriculturists for an explanation of the cause of the various scourges that fell upon their crops, perhaps their nearest approach to truth was in attributing certain types of injury to environmental factors such as soil and climatic or weather conditions. In certain observations of this kind the ancients were fairly accurate although, of course, they did not understand just why adverse weather conditions, for example, might cause a severe outbreak of one type of disease or another.

The history of plant disease control parallels, in a general way, the growth of knowledge of the nature and cause of plant disease. As long as man was ignorant of the etiology of plant diseases and tried to account for them on a supernatural basis, just so long did he propose many remedies, based upon superstition, which had absolutely no value when viewed in the light of our present knowledge of the causal agents. Many of these proposed remedies had to do with the propitiation of the

gods to whose wrath the maladies of plants were supposed to be due. As soon as man arrived at a rational understanding of the nature of disease he was in a position to think out logical means of disease control based on scientific knowledge rather than on ignorance and superstition.

**The science of plant pathology.** — In the earliest writings the references to plant diseases were very fragmentary and were scattered through the religious and historical writings of the Hebrews, Greeks and Romans. As time went on the accounts of plant diseases gradually came to be segregated in the writings on botany and agriculture. The growth of knowledge on this subject was very slow and it was not until the last half of the nineteenth century that a sufficient body of scientific knowledge about plant diseases had accumulated to make it possible to organize this subject as a separate branch of science. Plant pathology, as at present organized, is the science which treats of plant diseases. The technical name now in common use to designate this science is *phytopathology*, a term derived from the Greek, meaning, literally, a discourse on plant disease. At the present time plant pathology, or phytopathology, is a subject recognized by practically all colleges and universities either as a separate science or as a phase of botanical science.

**Eras in the history of phytopathology.** — While plant pathology as an organized science is very young, it had its beginnings in the earliest historical times. It will be of interest to trace briefly the growth of knowledge about plant diseases in so far as this information can be gleaned from the writings of past ages. Since the records of the existence of plant diseases and the notions held concerning them are necessarily available to us only in such historical writings as have come down to us through the ages, the eras into which the history of plant pathology naturally falls correspond very largely to the eras usually recognized in works on general history. For a brief survey of the rise and development of the science of phytopathology, then, we may consider its status during the various periods of ancient, medieval and modern times.

**The ancient era.** — This includes the period of time usually covered in writings on ancient history and extends from the dawn of history to 476 A.D. In so far as records of plant diseases are concerned, these are confined to the writings of the Hebrews in Asia and the Greeks and Romans in Europe. The contributions to our knowledge of plant diseases made by the Hebrew nation are very vague and fragmentary, consisting of the few references found in the Bible. The following quotations are characteristic of the Bible references to plant diseases and indicate how vague and indefinite were the ideas of such diseases prevailing at that time.

"If there be in the land famine, if there be pestilence, blasting, mildew, locust, or if there be caterpillar; . . . ." (I Kings 8 : 37).

"The Lord shall smite thee with a consumption, and with a fever, and with an inflammation, and with an extreme burning, and with the sword, and with blasting, and with mildew; . . . ." (Deuteronomy 28 : 32).

"I have smitten you with blasting and with mildew; . . . ." (Amos 4 : 9).

The Hebrew people invariably conceived of epidemics and plagues of whatever kind as manifestations of the displeasure of God visited upon the people for actions of theirs not in accord with His wishes.

Some of the early Greek philosophers were botanists and it is in such writings of these men as have survived that we find the most extensive accounts of plant diseases that have been gleaned from ancient history. **Aristotle** and **Theophrastus**, who lived some 300 years B.C., are the most renowned of these early Greek botanists although they were not the first Greeks to study and write about plants. In his "Landmarks of Botanical History," Greene (10)\* states that **Cleidemus** who lived prior to Aristotle's time was the first plant pathologist. The written works of Cleidemus have not survived and we know of his work only because of the fact that he was quoted by Aristotle and Theophrastus. He made observations on the diseases of plants, particularly of the fig-tree, the olive-tree and the vine. Theophrastus is generally referred to as the Father of Botany because he organized the subject better than anyone previous to his time had done and wrote voluminously of plants. He is said to have written over two hundred treatises on various subjects, only a few of which have been preserved. The chief one of these is his *Historia Plantarum* which deals with the botany of plants but in which are found many references to plant diseases.

The observations of Theophrastus indicate that even in that early day some of the notions held concerning plant diseases had a considerable element of truth although there was also much superstition mingled with these more rational ideas. The following quotations from Theophrastus (16) indicate the status of knowledge on plant diseases at that time and show that even the philosophers were not free from superstition.

† "The olive, in addition to having worms (which destroy the fig too

\* Arabic numerals in parentheses, as above, refer to references listed at the end of each chapter.

† Theophrastus. Enquiry into plants, and minor works on odours and weather signs. (English translation by Sir Arthur Hort.) Courtesy G. P. Putnam's Sons, Publishers, New York and London.

by breeding in it), produces also a knot (which some call a fungus, others a bark-blister), and it resembles the effect of sun-scorch. . . . The fig is also liable to scab, . . . ."

"The fig is also often a victim to rot and to *krados*."

"Scab chiefly occurs when there is not much rain after the rising of the Pleiad; if rain is abundant, the scab is washed off, . . . ."

"Generally speaking, cereals are more liable to rust than pulses, and among these barley is more liable to it than wheat; while of barleys some kinds are more liable than others. . . . Moreover the position and character of the land make no small difference in this respect; for lands which are exposed to the wind and elevated are not liable to rust, or less so, while those that lie low and are not exposed to wind are more so. And rust occurs chiefly at the full moon."

These brief statements are characteristic of the thought of the times. Some of the shrewd observations on the effect of such environmental factors as the lay of the land and the weather are good plant pathology, even today, but the strong hold which superstitious beliefs in the influence of the moon and stars had upon the minds of the people was not to be shaken off until modern times.

The contributions which the Romans made to plant pathology are found chiefly in the "*Natural History*" by Pliny who lived A.D. 23-79. This work consists of thirty-seven books and is largely a compilation made up of translations into Latin from the Greek writers, Aristotle, Theophrastus and others. Pliny's explanations of the cause of diseases were perhaps even more superstitious than those of the early Greeks. The Romans thoroughly believed in the influence of their various gods in bringing about epidemics of disease and tried to propitiate them by holding festivals and offering sacrifices.

**Plant pathology in medieval times.** — Beginning with the fall of the Roman Empire in 476 A.D. and continuing for approximately a thousand years, the world fell into decay as far as learning and the ancient civilization were concerned. During this period, known as the **Dark Ages**, no great contributions to history or literature in general were made and a large part of the ancient writings was lost or destroyed. Consequently this period is practically a blank so far as additions to the knowledge of plant diseases are concerned. But beginning about the fourteenth century there was a gradual **Revival of Learning** which started in Italy and gradually spread over central and western Europe. Universities sprang up in numerous places and many people devoted themselves to a study of the classics which had been preserved from ancient times. Thus the students of this **Renaissance** period had access to such botanical and agricultural writings of the Greeks and Romans

as had escaped destruction during the long centuries when learning slumbered. The revival of interest in agricultural writings gradually grew during the fifteenth, sixteenth and seventeenth centuries. Many records of plant diseases occur in the writings of the seventeenth century, especially, but there is not much that is new. Those who wrote were influenced in their opinions almost entirely by the philosophies and superstitions which they found expressed in the ancient writings to which they had access.

**The transition period (1700–1850 A.D.).** — With the beginning of the eighteenth century a decided increase of interest in plant diseases can be perceived. Many noted writers on botany and plant diseases lived at this time. There is exhibited in the writings of this period a strong trend toward organization of the available knowledge on plant diseases into some definite system. There was a decided tendency to classify diseases, and many writers set forth their views on the proper system of classification for plant diseases. Even at that early date opinions differed as to the basis of classification. (See Chapter III.) Tournefort, in 1705, classified diseases on the basis of cause. Zallinger, in 1773, used the symptoms of disease as a basis for his system of classification. In 1774 Fabricius (9) published an elaborate classification of plant diseases based entirely on cause. It must be understood that those who classified diseases on the basis of cause were struggling under the handicap of ignorance of the true cause of most of the diseases then known, especially the parasitic diseases. Nevertheless, these botanists, while busy classifying diseases, were also continually raising questions as to the nature and cause of disease. They were no longer satisfied with the explanations found in the old classics. During the latter part of the eighteenth century and the first half of the nineteenth, a new idea gradually took hold of the workers in this field. One of the most puzzling questions was concerned with the nature of the various morbid growths occurring on diseased plants, which we now know to be the result of fungous attack; such, for example, as occur in connection with the smut and rust diseases. Unger and others came to believe that such diseases were brought about by internal disturbances in the physiology of the plant and that the peculiar growths appearing were due to a kind of degeneration of tissues arising from within the plant. This idea has been referred to as the physiologic or *autogenetic* (21) theory of disease.

But during this time, especially the first half of the nineteenth century, still another idea was gradually taking hold among those who investigated plant diseases. The increasing use of the microscope in connection with the study of diseased tissues was leading some investi-



gators to question the authenticity of the *autogenetic* theory of the cause of disease. This was especially true of the fungous diseases. The idea that the spores and other fungous structures found in or on disease lesions might be separate and distinct organisms living upon the host plant and causing the diseased condition gradually grew until by the middle of the nineteenth century the truth of this assumption was definitely established.

**The modern era** (1850 to the present). — The discovery of the pathogenic nature of fungi led to an intensive study of these organisms as the cause of plant diseases and during the latter half of the nineteenth century "plant pathology" became practically synonymous with "fungous diseases of plants" (7). In other words this may be called the period of applied mycology. Whetzel (21) terms the period from 1853 to 1883 the *pathogenetic* period, that is, the period during which the theory that plant diseases are caused by pathogenic organisms superseded the autogenetic theory which had held sway during the preceding period.

In reviewing the history of phytopathology in Europe during the years 1850 to 1900, one becomes aware that the list of workers in this field is a long one. Some of the more familiar names found in the literature of plant pathology outside the United States are listed below by countries. Germany: De Bary, Kuhn, Frank, Brefeld, Hartig, Schroeter, Sorauer, von Tubeuf, Kirchner, Winter and Klebahn — France: Millardet, Prillieux and Delacroix — Italy: Comes, Berlese and Savastano — Sweden: Eriksson — Denmark: Rostrup, Ravn and Jensen — Russia: Woronin — Holland: Ritzema Bos. In England the most outstanding workers during this period are Berkeley and Ward, while in Australia McAlpine deserves mention.

Anton de Bary stands out as one of the greatest workers at the beginning of the modern era because his extensive work on the life histories of the fungi contributed a great deal toward a better understanding of the true nature of these organisms and placed the theory of the pathogenetic nature of plant diseases on a firmer basis. Among his best known works are extensive investigations on "*Die Brand Pilze*" (1) in which he definitely established the nature of the smut and rust fungi. One of his best remembered pieces of work in connection with rusts is that in which he proved the relationship between the two stages of the stem rust of wheat, the one stage on the barberry and the other on wheat. Much of his work on fungi is summarized in "*Morphologie und Biologie der Pilze*, etc." (3). De Bary, however, was a mycologist rather than a plant pathologist, but in spite of this fact his work was of inestimable value to the plant pathologists of the early part of the modern era.

Julius Kühn (12) wrote the first modern textbook of plant pathology. This was entitled "*Die Krankheiten der Kulturgewächse, ihre Ursachen und ihre Verhütung*," and was published in 1858. It was the first textbook on plant pathology to embody the new knowledge concerning the fundamental relation of fungi, as causal agents, to plant diseases. The book consists of two parts. Part One is given over to a discussion of general topics such as the nature, types and causes of disease; while Part Two consists of a discussion of investigations on various specific diseases known at that time. Robert Hartig is recognized for his textbook on the diseases of trees. Brefeld published many studies on the fungi, beginning about 1872 and continuing over into the early part of the twentieth century. His work on the grain smuts is of particular interest. Frank published a textbook on plant pathology in 1880. During his life he wrote eleven books on various phases of botany including plant physiology and plant pathology. Sorauer (15) is noted chiefly for his "*Handbuch der Pflanzenkrankheiten*," the first edition of which, a single volume, came out in 1874. A second edition in two volumes was published in 1886, and a third edition in three volumes was completed in 1913. Volume One of the third edition, which treats of non-parasitic diseases, has been translated into English. A fourth edition of Sorauer's manual, in five volumes, was brought out in 1921-1925 by Graebner, Lindau and Reh. In 1895 Tubeuf published a textbook on plant diseases caused by cryptogamic parasites. This book also has been translated into English.

In France Millardet is noted for his discovery, in 1882, of bordeaux mixture as a fungicide for the downy-mildew of grape. Other French plant pathologists of note are Delacroix and Prillieux, each of whom published a two-volume work on plant diseases. In Denmark the leading plant pathologist during the formative period of modern phytopathology was Rostrup whose works on plant diseases appeared after 1870, although he had published other botanical works previous to that date. Eriksson is Sweden's outstanding phytopathologist. With Henning he made very important contributions to our knowledge of the cereal rusts. He first discovered biologic races or specialized forms within morphologic species of rusts. He is also noted for his "mycoplasma theory" which he advanced to account for the perpetuation of the rust fungus and the sudden outbreak of rust epidemics. Woronin, a Russian, is noted chiefly for his work on the club-root of cabbage. Cavara of Italy is said to have been the first to isolate the crown-gall organism. Savastano, another Italian, is noted for his work on the olive knot as well as other pathological investigations. In England, Berkeley was the chief figure in plant pathology at the beginning of this era. He was first of

all a mycologist but took an especial interest in the diseases caused by fungi, and many of his publications are pathological in nature. Ward, the greatest English plant pathologist, began his career in 1881. His first work was an investigation of a coffee disease in Ceylon. Later he wrote much on plant diseases, among his greater efforts being his textbook, "Disease in Plants," published in 1901. McAlpine in Australia is known by his works entitled, "The Smuts of Australia," "The Rusts of Australia," and "Bitter-pit Investigations."

**Modern plant pathology in America.** — It is thus seen that the science of plant pathology had its inception in ancient times in the civilized nations inhabiting the shores of the Mediterranean Sea. It is true that the beginnings of the science were not very scientific but the seeds were sown there, especially by the Greeks and Romans, which, after the lapse of many centuries, were to blossom out during the nineteenth century in the educational centers of Europe into a real science of phytopathology. It was with this background, then, that plant pathology was transplanted to the United States during the second half of the nineteenth century. In this country the first attempts at organizing a separate science of plant pathology occurred between 1870 and 1880. In 1873 instruction in plant diseases was first given in connection with botany by Burrill at the University of Illinois. In 1875 a special course in plant pathology as such was offered for the first time by Farlow at Harvard University.

Plant pathology has developed in the United States chiefly under two separate agencies, namely, (a) the United States Department of Agriculture and (b) the Agricultural Colleges and Experiment Stations of the several states (19). These two agencies, however, have worked in more or less close coöperation. The Federal Department of Agriculture carries on work in plant pathology chiefly under the various offices of the Bureau of Plant Industry. The first organized effort was initiated in 1885 with the organization of a Section of Mycology in the Division of Botany of the U. S. Department of Agriculture. Since that time the work has developed rapidly until today there are more than a dozen different offices, chiefly in the Department of Agriculture under which plant pathological investigations are carried on. In addition to the workers in the various offices at Washington, D. C., field stations are maintained at many points throughout the country where investigations of particular diseases can be carried on to advantage. In some cases these field stations are located at a state experiment station where the work is done in coöperation with the state stations. In other cases stations are established at other vantage points and not in direct connection with the state experiment stations.

The State Agricultural Experiment Stations came into being as a result of the Hatch Act of 1887 in accordance with which the Federal Government appropriates \$15,000 per year to aid each state in carrying on scientific investigations in agriculture. The Adams Act of 1906 and the Purnell bill of 1925 provided further Federal aid to the various states for this work. In addition to these Federal funds, each state makes its own appropriations for experiment station work. Along with the other agricultural investigations, practically every state maintains one or more workers in plant pathology; thus the growth of plant pathology in the state experiment stations has kept pace with its development in the United States Department of Agriculture. The agricultural colleges also offer courses in plant pathology so that all agricultural students may have scientific and practical instruction on plant diseases and their control.

**Summary of important events and developments in the history of phytopathology.** — In summarizing the chief points in this historical account of plant pathology an attempt has been made to select and list some of the more important contributions which have been made from time to time toward the upbuilding of the science as we know it today. No claim is made that this list includes all worthy items, but it is felt that such items as are listed here may be considered milestones along the road toward a better and more complete knowledge of the things necessary to make this science of most service to humanity. The order of listing the items is, in general, chronological, although in some cases where the item is more in the nature of an epoch or a gradual development no definite dates can be assigned.

1. Probably the first definite contribution to our knowledge of plant diseases was made by Theophrastus, who lived about 300 B.C., and has come down to posterity in his work, "*Historia Plantarum*."

2. Pliny's "Natural History" (first century A.D.) is the next great work which contains information of value on plant diseases.

3. The discovery of bacteria by Loewenhoeck in 1683 was ultimately to wield a great influence in the field of plant pathology because of the discovery of bacterial diseases in plants at a much later date.

4. The systematic classification of plant diseases by Zallinger and others during the eighteenth century. While this work was more or less superficial, nevertheless it was important because it brought about an organization of such knowledge as was available at that time and paved the way for rapid advancement later.

5. The evolution of the autogenetic theory of the cause of plant disease, which crystallized under the leadership of Unger during the first half of the nineteenth century.

6. The use of copper sulfate as a seed treatment for grain smut by Prévost in France (1807).

7. The first use of sulfur as a fungicide by Robertson (1821). It must be remembered that these early attempts to control plant diseases by the use of copper and sulfur were entirely empirical since the true nature of fungi was not known at that time. Nevertheless, even at this early date, it is known that some workers began to suspect the true nature of fungous spores and these conjectures were soon to take definite form in the epoch-making discoveries to follow.

8. The final proof of the parasitism of fungi established by De Bary and others near the middle of the nineteenth century.

9. In 1864 De Bary published the results of his work on the stem rust of wheat in which he established the heteroecious nature of the rust fungus, proving that the telial stage on wheat and the aecial stage on barberry are but two phases in the life history of the rust fungus. This discovery has been of inestimable value to all succeeding students of the rust fungi.

10. The founding of the science of bacteriology by Pasteur (1860-1864).

11. Plant pathology first taught in the United States (1873). Burrill of the University of Illinois is said to have given the first instruction in this subject as a part of the regular courses in botany.

12. The first independent course in plant pathology was organized by Farlow at Harvard in 1875.

13. The first definite proof of a bacterial disease in plants. Burrill of Illinois determined the cause of fire-blight of pears and apples to be a bacterium (1879-1880).

14. The development of the "plate method" of isolating bacteria and fungi by Koch (1881). The final step in this innovation resulted in the use of agar in Petri dishes as used today.

15. The discovery of bordeaux mixture as a fungicide for use as a spray by Millardet (1882). It was accidentally discovered that a mixture of lime and copper sulfate sprinkled on grape vines to deter pilfering of grapes was a good remedy for the downy-mildew of the vine.

16. The establishment of a Section of Mycology in the Division of Botany, U. S. Department of Agriculture (1885).

17. Proof of the infectious nature of mosaic by Mayer (1886). He demonstrated that tobacco mosaic could be transmitted by inoculating a healthy plant with juice expressed from a diseased plant.

18. Development of the hot-water method of seed treatment for cereal smuts by Jensen of Denmark (1887). This method in modified form is still recommended for loose smut of wheat.

19. The passage of the Hatch Act (1887). This paved the way for the establishment of the state experiment stations (1888).

20. The use of formaldehyde as a disinfectant. Trillat is said (12) to have first introduced it in 1888. Orton gives Geüther credit for first using formaldehyde as a seed disinfectant in 1895. Bolley introduced it for bunt and other smuts in 1897, and Arthur used it for potato-scab control in 1897. The use of hot formaldehyde for potato-seed treatment was developed by Melhus and others (1918-1920).

21. The discovery of biologic races of species of rust fungi by Eriks-son (1894).

22. Passage of the Adams Act (1906). This bill provided additional funds for experimental work at the state stations, and plant pathological investigations profited thereby.

23. The adaptation of lime-sulfur spray for the control of apple-scab by Cordley (1906-1908).

24. The organization of the American Phytopathological Society (1909). This served as a source of stimulation and inspiration for those interested in plant pathology as a profession.

25. The passage by Congress of the Federal Plant Quarantine Act (1912). This was the beginning of the attempt to exclude plant diseases by means of legal embargoes placed against the importation of diseased plants or plant parts.

26. The substitution of fine dusts for liquid sprays in the control of certain diseases (1910 to present). Both the sulfur and the copper fungicides are now available in this form. There are many advantages in the use of dusts over liquid sprays and they have proved their worth in many cases. On the other hand there are many cases in which dusting has never proved as successful as spraying.

27. During the last ten or fifteen years, and especially since 1920, the virus diseases have come into great prominence and have received an increasing amount of attention from plant pathologists, with the result that our knowledge of them has been materially advanced.

28. During the first quarter of the twentieth century and continuing to the present time, few if any phases of plant pathology have received more attention than the problem of resistance and immunity. Research in this field has centered around the selection and breeding of resistant or immune varieties, the genetics connected therewith, and the basis of immunity in plants.

29. The substitution of copper carbonate dust for copper sulfate and formaldehyde dips in the treatment of seed wheat for bunt (1915 to present). This seed disinfectant has been in use in Australia since 1915, but in the United States only since about 1921.

## REVIEW QUESTIONS

1. Where are the earliest writings on plant diseases found?
2. To what were plant diseases attributed in the earliest historical times?
3. Name two of the most renowned of the early Greek botanists.
4. Who is credited with being the first plant pathologist?
5. What is the literal meaning of the term "phytopathology"?
6. What early Roman writer contributed most to our knowledge of plants and plant diseases?
7. Discuss the state of, and the contributions to, knowledge of plant diseases during the middle ages.
8. When did Tournefort, Zallinger and Fabricius live, and what did each contribute to our knowledge of plant diseases?
9. What is meant by the "autogenetic" theory of plant disease? The "pathogenetic" theory?
10. During what period of time did each of the above theories come into prominence?
11. Give a list of the greatest plant pathologists of the last half of the nineteenth century. Mention the native country of each.
12. What is the difference between "plant pathology" and "mycology"?
13. For what is De Bary noted? Kühn? Brefeld? Hartig? Millardet? Sorauer? Eriksson?
14. Discuss the beginnings of plant pathology in the United States.
15. Under what two chief agencies has the science of plant pathology developed in the United States during the last fifty years?
16. Mention twenty-five or more important events or landmarks in the history of plant pathology.

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## CHAPTER II

### MODERN PLANT PATHOLOGY

In the preceding chapter it was pointed out that the development of the science of plant pathology was very closely related to, and parallel with, the rise and growth of botanical knowledge. The modern plant pathologist is cognizant of his constant dependence upon the botanical sciences in his investigations of plant diseases. Some of the phases of botany, a knowledge of which is more or less indispensable to the plant pathologist, are mycology, morphology, anatomy and histology, physiology, cytology, plant genetics, ecology and bacteriology. The botanical sciences are not, however, the only ones concerned in a study of plant diseases. Chemistry and physics, especially the former, are of importance. A knowledge of certain phases of chemistry is especially necessary in such studies as the physiology of the fungi. Zoölogy, too, should be mentioned in this connection since certain types of animal parasites come within the field of plant pathology. A somewhat more detailed discussion of the dependence of plant pathology upon these branches of science, especially the various phases of botany, should be of interest here.

**Mycological relations.** — Mycology may be defined as the science of fungi. In the early stages of its development the science of plant pathology dealt almost entirely with those diseases which are caused by fungi, and consequently the term "plant pathology" was practically synonymous with the term "fungous diseases of plants." While this is no longer true and many types of trouble other than the strictly fungous diseases are legitimately considered within the field of plant pathology, yet the fungi still play a very important part as causal agents of many of our most serious plant diseases. Consequently some knowledge of mycology is indispensable to the plant pathologist who would know his field thoroughly.

The fungi may be considered from various standpoints, namely: the taxonomic, the morphologic and the physiologic (1). In studying the causal fungus of a disease, it is, of course, desirable to know its systematic classification, but merely being able to identify the organism is not enough. Its morphology and physiology should also be known. The structure of its vegetative phase and its various spore forms should be studied, and its complete life history worked out. In studying the

physiological aspects, attention should be turned to the conditions under which spore germination and infection will occur; the conditions most favorable for rapid invasion of the host; the conditions conducive to the development of the different fruiting stages; and conversely, the conditions unfavorable to any of these activities.

**Anatomical and histological effects.** — The effect of most plant diseases, whether parasitic or non-parasitic, is to alter in one way or another the anatomical or histological structure of the diseased plant. The changes wrought in the structure of the plant or plant parts may be most diverse. These modifications or alterations in the host structure are usually included in the term "symptoms" of disease. Essentially all kinds of symptoms may be included under the three general types, namely, the necrotic, the hypoplastic or the hyperplastic diseases. The first of these three types of symptoms is characterized by a killing of the affected tissues. Necrosis is the chief anatomical or histological effect manifest in a very large number of our most important plant diseases. All the rots, cankers and leaf-spot diseases are examples of the necrotic or killing effect of plant diseases. The hypoplastic diseases result in a dwarfing or underdevelopment of the plant or plant parts, and the hyperplastic type is illustrated by such overgrowths as club-root of cabbage and crown-gall. Any of these different types of structural modifications may affect only a few cells, or even a single cell, on the one hand, or they may extend to larger areas, sometimes including a whole organ or even the entire plant. All of these effects of whatever type are included under the term "pathological anatomy or histology."

**Physiological relations.** — The relation of plant physiology to plant pathology may well be considered under two heads, namely, in connection with the non-parasitic or so-called physiological diseases on the one hand; and in relation to the parasitic diseases, on the other hand. In considering the non-parasitic diseases, as for example, bitter-pit of apple, internal brown spot of potato, point-rot of tomato, or apple-scald, the problem resolves itself very largely, if not entirely, into one of the physiology of the diseased plant in relation to its environment. The cause of the first three diseases mentioned above is probably a question of unbalanced water relations, while in case of the apple-scald, it is largely a respiratory problem.

The parasitic diseases offer a large field for the consideration of the physiology of both the host and the parasite. It is undoubtedly true that the physiological condition of the host before invasion has a great deal to do with its susceptibility to attack. It is equally true that the invasion by a parasite may profoundly influence the physiological behavior of the host. But while we are considering the physiological

reactions of the host either before or after infection by a parasite, we must not forget that the physiological reactions of the parasite itself are important factors to consider. The existence of resistance and susceptibility to disease in plants furnishes a good example of the fine adjustment of these inter-reactions occurring between host and parasite. If, as is generally believed, the problem of immunity is largely a physiological one, it means that, in case of a variety or individual which is immune to the attack of a certain parasite, not only are the physiological reactions of the host incompatible with those of the parasite, but *vice versa*. This is well illustrated by the great differences in varietal susceptibility of wheats to stem rust. In this case the rust fungus itself exhibits many different strains or biologic races not all of which can attack any particular variety of wheat with equal facility. Here not only does the physiological reaction of a particular variety of wheat differ from that of other varieties with reference to a particular rust strain, but the reaction of one strain of the rust fungus differs from that of other strains with reference to any particular variety of wheat. All this, of course, is based upon the supposition that resistance and susceptibility are due to physiological factors rather than to morphologic or histologic structure.

**Inter-relationship of physiology and anatomy or morphology.** — It is a generally recognized fact that a close relationship exists between structure and function in normal plants. This relationship is also evident in diseased conditions. An alteration in the structure of a plant due to an attack of a parasite may be followed by a change in the functional processes, which in turn may be manifested by further structural changes. This is well illustrated in the Rhizoctonia disease of potatoes. Here the fungus (*Corticium vagum*) may kill the cortical tissues to such an extent as to girdle the stem. This interferes with the normal transfer of carbohydrates through the phloem to the usual place of storage in underground tubers, and results in storage above ground, giving rise to further structural changes as exhibited in the formation of aerial tubers.

**Cytology and plant genetics.** — Cytology, or the study of the cell, plays a part in many investigations in plant pathology. In some plant diseases, the parasite lives in intimate relationship with the living protoplast of the host cell. Certain diseases caused by species of slime molds and by species belonging to the order Chytridiales illustrate this fact. The organism causing club-root of cabbage lives as a plasmodium in close association with the host protoplasm in the cells of the diseased cabbage roots. A correct understanding of the relation of parasite to host necessitates a careful cytological study of the affected tissues.

The laws of heredity are based upon certain cytological facts, and the breeding of plants immune to disease must necessarily depend upon a knowledge of these cell phenomena and the laws governing the inheritance of the characters, resistance and susceptibility. The subject of resistance and immunity will be discussed at greater length in Chapter XI.

**Ecological relations.** — Ecology treats of the relation of plants to their environment. Plant diseases, both parasitic and non-parasitic, are very intimately affected by environmental factors such as soil and climatic or weather conditions. These factors affect the host directly in the case of the non-parasitic diseases; and in the case of the parasitic diseases they exert a profound influence upon the activities of the causal organism, in addition to the direct effect they have upon the host. In many cases, weather conditions which induce a healthy, vigorous growth in the host plant make it much more liable to attack by plant-disease-producing parasites. Other diseases attack plants more readily when the plants are not in a healthy condition. Environmental conditions which are not conducive to healthy growth of the plant would make it more susceptible to attack by such diseases. Most plant-disease organisms are very sensitive to the ecological factors of temperature and humidity. Unless these factors are favorable the organisms do not infect and develop to a serious extent. If the weather conditions are favorable an epidemic is apt to occur. For further discussion of the relation of plant diseases to their environment, see Chapter V.

**Bacteriology.** — Many plant diseases are caused by bacteria, and consequently a knowledge of these organisms is necessary in order to combat this type of disease. Bacteriology is a recent offspring of botany, but bacteriologists have developed an extensive science and technique which are very useful to the plant pathologist. The most extensive works on bacteriology as related especially to plant pathology are those of Erwin F. Smith (2, 3).

**Zoölogy.** — By far the greater part of the injuries to plants caused by animal life is due to the depredations of insects, and is properly handled by entomologists. However, there is one group of animals which causes enormous damage to plants, but which, by common consent, is turned over to the plant pathologist rather than to the entomologist or zoölogist for investigation. This group consists of the eelworms or nematodes. These minute worms cause a variety of injuries to plants. Their depredations are so widespread and the problems connected with the investigation of this group of plant pests are so extensive, that there has arisen a distinct branch of plant pathology known as nematology, the exclusive field of which is the investigation of plant diseases caused by eelworms.

**Entomology.** — The close relationship which insects bear to certain plant diseases makes it desirable that the plant pathologist have some knowledge of insects. Many plant diseases may be disseminated by insects and certain diseases are transmitted in no other way. Certain of the bacterial diseases, such as fire-blight, and most, if not all, of the virus diseases depend largely or entirely upon insects for dissemination. Hence the worker who would investigate these diseases must either know something of the identity and habits of the insects concerned or work in close coöperation with an entomologist. (For a more detailed treatment of the relation of insects to plant diseases see Chapter XII.)

#### REVIEW QUESTIONS

1. To which one of the natural sciences is plant pathology most closely related?
2. Name the different phases of botanical science, a knowledge of which is indispensable to the plant pathologist. Explain how each is useful.
3. Define each of the phases of botany implied in question 2.
4. Name some other sciences in addition to botany, a knowledge of which is useful to the plant pathologist.

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## CHAPTER III

### DEFINITION, SYMPTOMS AND CLASSIFICATION OF PLANT DISEASES

In order that we may have a more definite conception of some of the items of the subject-matter of plant pathology it is desirable that we attempt to formulate some kind of a definition or description of what constitutes disease in plants, and that we be able to recognize the symptoms which indicate an unhealthy condition. Also the working out of a satisfactory system of classification for the various maladies which affect plants will be of value to us in formulating an orderly conception of the field covered in this course. The end aimed at in attempting to define plant disease is not to set up a formal definition to be mechanically memorized, but rather to stimulate thought on the subject in order that we may actually have a clearer conception of plant disease than we would get by merely committing to memory a formal definition. Symptoms are the expression of disease in plants, and consequently a knowledge of the different symptoms exhibited by various diseases is necessary for their correct identification.

#### Definition of Plant Disease

In the works of various writers on plant pathological subjects we find many attempts to define plant disease. Some of these definitions are brief and concise while others are lengthy and more or less involved. It will be of interest to analyze some of them and learn to what extent they measure up to our present conception of what disease in plants means. Perhaps this very act of analysis will enable us to formulate a more concise and workable definition of our own.

Hartig (2) gives the following definition:

\* "I do not regard the investigation of the phenomena of mere sickness as the task of pathology. It is only when the sickly condition leads to the death of some part of the plant that we speak of actual disease."

It is very questionable whether this definition would include such diseases as apple-scab, crown-gall or witches' broom, which though they

\* Hartig, Robert. Diseases of Trees. The Macmillan Company, New York. 1894. Reprinted by permission.

may not necessarily result in the death of the plant or any part of it, yet certainly are considered diseases in our present-day accepted conception of plant disease.

Ward (8) gives essentially the same definition, though couched in different words. His definition follows:

\* "Disease . . . may be said to be variation of functions in directions, or to extents, which threaten the life of the plant, the normal in all cases being the state of the plant characteristic of the species."

The following definition by Morse and Lewis (4) is very brief but at the same time very inclusive:

"Disease in plants includes the effect of every unfavorable factor entering into the life of the plant."

It would be difficult to think of any type or phase of disease which would be excluded by the above definition. Indeed it is probable that this definition would include conditions which we would not ordinarily consider diseases.

Sorauer (5) undertakes to distinguish between those diseases which actually kill the plant and those which damage it in one way or another without resulting in its death. He uses considerable space in expressing his conception of plant disease, but his definition is interesting and worthy of careful thought.

† "If we call 'sick' only those cases in which the organism undergoes such a disturbance in its function that its existence seems threatened, we will be in a dilemma when we consider the changing developmental forms of our cultivated plants, for we will then discover that the above explanation is insufficient . . . .

" . . . In treating questions of disease, we shall have to follow two lines of work. We must naturally first keep the organism's aim in sight. And this aim, which the organism derives from its very origin, is to live, and in fact to live as long as possible . . . In following the second line, the aim of cultivation, developed from the relation of the plants to human needs, is an added important factor. These conditions of the vegetable organism opposing our cultural endeavors will be combated as inexpedient. But such conditions need in no way threaten the existence of the individual and therefore, according to the above explanation, are not diseases, yet they belong to the province of the pathologist as disturbances which must be considered and overcome . . . .

"The consideration of the cultural needs forces us to the following

\* Ward, H. Marshall. *Disease in Plants*. The Macmillan Company, New York. 1901. Reprinted by permission.

† Sorauer, Paul. *Manual of Plant Diseases*, Vol. I. English translation by Dorrance. The Record Press, Wilkes-Barre, Pa. 1914-1920.

division of our subject; first of all, we will have to consider all cases which threaten the individual aim of the organism, i.e. its longest possible life; — these are *absolute diseases*. Then we must discuss the disturbances which the momentary cultural aim experiences and which we term *relative diseases*. These relative diseases may vary since what cultivation considers worth striving for today may be neglected tomorrow."

It is readily apparent that the above definition includes not only those diseases which threaten the life of the organism, but also those which lower the value of a crop without completely destroying it.

The literature of plant pathology contains many other attempts to define plant disease but it would not be profitable to use more space to discuss all of them here. Perhaps the best features of all these definitions could be combined to make an all-inclusive definition the essential points of which would be as follows: (a) Disease is a disturbance of, or a deviation from, either the normal structure, or the normal function, or both. (b) It may affect the whole plant or only a part of it. (c) It may threaten the life of the affected plant or plant part, or only lessen its economic value.

### Symptoms and Signs of Disease

The signs and symptoms of disease are those marks or evidences which indicate a diseased condition in the plant. Most writers do not distinguish sharply between symptoms and signs, but for the sake of exactness perhaps it is better that we do so. Whetzel (11), for example, insists that a distinction should be made in the use of the two terms. He defines symptoms as \* "those expressions by the suspect of a pathologic condition by which a diseased plant is distinguished from a healthy one," and signs as "incidental or experimental evidences of disease as distinguished from pathological evidences. The usual signs of disease are the structures of the pathogene produced in or on the lesions." For practical purposes of identification the "signs" are sometimes just as useful as the symptoms of the disease, or perhaps even more so in certain cases. For the layman or the student who is pursuing a brief course in plant pathology simply because of the value it will have for him in the identification and control of plant diseases, probably no practical purpose is served in differentiating between signs and symptoms.

**Symptoms.** — The symptoms of plant diseases, interpreted in the strict sense as expressions of pathologic conditions, fall into three general classes, namely, the necrotic, the hypoplastic, and the hyperplastic

\* Whetzel, H. H. Laboratory Outlines in Plant Pathology. 1925. Courtesy W. B. Saunders Company.



types. The necrotic type includes all those symptoms which are manifestations of necrosis or death of the affected tissues. The hypoplastic type is the result of dwarfing, stunting or underdevelopment of the plant or some part of it. The hyperplastic type is represented by overgrowths of all sorts. There are many different special manifestations of each general type of symptom. A brief description of the more important special symptoms classed under each of the three general types, will aid in forming a more definite conception of the various pathologic conditions to be encountered in diseased plants, and enable the student to recognize more readily the specific symptoms by which particular diseases are to be identified.

*Necrosis.* — The chief kinds of necrotic symptoms, — that is, symptoms resulting from the death of tissues, — are rot, canker, blight, wilt, damping-off, spot, streak, burn and shot-hole. Rot is a condition resulting when the cells, wall and content, are broken down and more or less consumed by enzymes secreted by the attacking organism. In some cases only the middle lamellae are digested, thus permitting the cells to separate and the tissues to disintegrate. Rots in fruits and vegetables are familiar to everyone (Figs. 66, 75). The rotted tissue may be soft or hard, dry or wet, and either odorless or possessed of offensive odor. The color of the rotted tissue also varies. The different types of wood rot or decay which are very common in forest and orchard trees, and in manufactured lumber, are also classed here. Canker is the result of the death of areas in the cortex. The cankers may be of any shape and size and may be superficial or penetrate to the cambium layer. In the former case only the outer layers of cortex are killed and no great damage is done. In case the injury extends to the cambium layer the dead bark usually sloughs off after a year or two, thus exposing the wood (Fig. 39). Blight is a somewhat less definite term than rot or canker. The term is generally used to designate a condition in which a rather conspicuous dying down of foliage or twigs occurs. In some cases only parts of the leaves die while in others whole leaves or all the leaves on a twig, or even a whole plant may be involved (Fig. 57). The late blight of potato is an example of a disease which may affect only parts of leaflets at times and again may kill the whole plant. In fire-blight of pears one of the conspicuous and most characteristic symptoms is the blighting of twigs where all of the leaves on the twig die, dry up and turn almost black. Wilt, as the name suggests, consists of a wilting of the plant and is the result either of the blocking of the water-transporting channels in the stem or of some injury to the absorbing system which lowers the rate of intake of water below that of the outgo due to transpiration. Damping-off refers to the rotting of

the stems of seedlings at the surface of the ground in such manner that the seedlings suddenly topple over. Spot is a term used in referring to small dead areas on various parts of plants, usually on leaves and fruits. Leaf-spots are very common symptoms of a number of plant diseases (Figs. 93, 160). They are usually distinguishable from blight by the fact that leaf-spot diseases seldom kill the entire leaf, while the blights frequently kill whole leaves or plants. Streak is a type of necrosis that results in fine dark-colored lines running over the leaf surface. These lines sometimes occur in potato tubers. When the lines run together to form a network this is sometimes termed net-necrosis. Burn is a necrotic condition of leaves or fruits due to sunburn or other causes. Shot-hole results if the dead areas in spotted leaves fall out, leaving small holes through the leaves as if riddled by shot.

*Hyperplasia.* — The hyperplastic symptoms are the result of overgrowths, such as gall, witches' broom, hairy-root, curl and scab. Galls are overgrowths which result when parasites in the plant stimulate the cells to abnormal activity instead of killing them as in the necrotic type of disease. In response to the stimulus the cells may react in one or both of two ways. They may divide rapidly and thus multiply the number of cells enormously above the normal, or individual cells may increase greatly in size. The ultimate effect of these abnormal activities on the part of the diseased cells is to produce large masses of tissue which are called galls (Fig. 20). Witches' brooms are formed when large numbers of adventitious buds are produced and grow out into shoots, thus giving rise to a dense bushy growth resembling a broom made of twigs (Fig. 65). Hairy-root is a term applied to a condition on the roots of trees resembling the witches' brooms above ground. Dense bunchy growths of fine roots sometimes appear, especially as a form of the crown-gall disease where they usually grow out from a slight gall-like swelling on the tap root not far below the surface of the ground (Fig. 23). Curl may sometimes be the result of hyperplasia and at other times it may result from a dwarfing rather than an overgrowth. In peach leaf-curl the leaf blade is greatly thickened and overgrown in proportion to the mid-rib, thus giving rise to great curling or distortion of the leaf blade (Fig. 62). In diseases such as common potato-scab there is an excessive formation of corky tissue over the surface of the diseased areas (Fig. 176).

*Hypoplasia.* — The hypoplastic symptoms are the manifestations of a slowing-down or dwarfing effect on the part of the causal agent. Chlorosis is also the expression of an under-development on the part of the chlorophyll apparatus of the plant. Both dwarfing and chlorosis are striking symptoms of some of the virus diseases (see Chapter XXIV).

They are also symptoms of many of the parasitic diseases. A few examples of the latter are the production of "little potatoes" by the *Rhizoctonia* disease, the dwarfing of the shoots and leaves of apple trees by the powdery mildew and a similar dwarfing of the shoots of grape vines by the downy-mildew fungus.

**Signs.** — The signs of disease as distinguished from symptoms include all the structures of the pathogene, whether vegetative or fruiting, which can be observed while making a diagnosis. These include such growths as have given rise to the general terms mold, mildew, rust, smut, mycelium, ooze, and the more specialized structures such as perithecia, apothecia, pycnidia, acervuli, aecia, uredinia, telia, sporophores, sclerotia, conidiophores, and conidia. The presence of these structures is a sign of fungous infection, and the particular kind of fruiting body found is an indication of the identity of the causal organism. Students who have had a course in elementary botany should recognize most of the structures mentioned above. Illustrations of the various forms will be found in Part II of this text as follows: perithecium (Figs. 84, 90); apothecium (Figs. 69, 78); pycnidium (Figs. 160, 166); acervulus (Figs. 76, 172); aecium (Figs. 126, 127); uredinium (Figs. 124, 128); telium (Figs. 125, 129); sporophore (Fig. 151); sclerotium (Fig. 139); conidiophore (Figs. 47, 71); conidium (Figs. 51, 76).

### Classification of Plant Diseases

A great many different ways of classifying plant diseases have been suggested by various writers in the past. In the early history of the science dealing with plant diseases the field belonging exclusively to plant pathology was not clearly marked. The early writers sometimes included among plant diseases, troubles that now belong to other fields of science, for example, entomology. A chronological arrangement of a series of examples of systems of classification chosen from the different periods in the history of plant pathology would, in a way, illustrate the historical growth of the subject. Such a study would show that in the beginning the field was large and indefinite but that there has been a gradual narrowing down of the field and a tendency to define carefully and classify the subject-matter of plant pathology.

There are several different bases on which plant diseases may be classified, namely, on basis of *cause*, on basis of *symptoms*, and on basis of *host*. The thing which determines which basis one would use in classifying plant diseases depends upon the phase of the subject in which one is chiefly interested. If the interest centers largely in the etiology of plant diseases, the logical basis on which to classify them would be that of the causal agent. If the gaining of familiarity with the effects

of the disease on the host, either for the sake of the knowledge or as an aid in identifying plant diseases, were the chief object in view, then one would be inclined to use the symptomologic basis of classification. But if one had already become familiar with the different kinds of causal agents and with the different types of symptoms encountered in plant disease studies, and if one were interested mainly in the diseases of certain crops, as potatoes, wheat, or apples, then it might prove convenient to group diseases under their respective hosts. Historically, most systems of classification for plant diseases have been based on cause, although there are some notable exceptions to this rule. It will be of interest now to examine and analyze a few different systems of classification chosen from different historical periods as well as to represent the different bases of classification. In doing this we shall familiarize ourselves with the development of the subject-matter of plant pathology sufficiently to enable us to work out a suitable classification of the plant diseases with which plant pathologists have to deal today.

**Classification of plant diseases on basis of cause.** — One of the earliest attempts to classify plant diseases was made by Tournefort (10) in 1705. He made two main classes, namely, (a) diseases due to external causes, and (b) diseases due to internal causes. Reference to Chapter I will show that knowledge of the true cause of plant diseases was non-existent at that date, at least in so far as parasitic diseases are concerned. His class due to external causes would probably correspond to the group we now know as non-parasitic diseases, that is, diseases largely due to environmental factors such as weather conditions. His class due to internal causes was based largely upon the superstitious notions handed down from ancient and medieval times.

In 1774 Fabricius published an elaborate system of classification based partly on the causal agent and partly on symptoms. He included six main classes in his outline as follows: (a) Rendering unproductive; (b) Wasting; (c) Discharging; (d) Decaying; (e) Injury; (f) Extraneous. Many subdivisions, based largely on cause, were included under these main heads, as, for example, diseases due to cold; those due to drouth; those due to lack of light; those due to unsuitable soil; those due to external injury; those due to parasites; etc.

Coming down to modern times (1894) we find this classification in Hartig's "Diseases of Trees" (2): (a) diseases induced by Phanerogams (seed plants); (b) diseases induced by Cryptogams (seedless plants); (c) wounds; (d) diseases due to unfavorable conditions of the soil; (e) diseases due to unfavorable atmospheric conditions.

In 1901 Ward (8) proposed the following two classes to include all plant diseases: (a) diseases due to the non-living environment; and

(b) diseases induced by the activities of living organisms. It will be noticed that Ward's two classes include all of Hartig's five classes with the possible exception of wounds. Hartig's classes (a) and (b) fall under Ward's class due to living organisms, and his classes (d) and (e) together correspond to Ward's class due to the non-living environment. Even Hartig's third class, wounds, are caused either by living or non-living agents; therefore Hartig's entire five classes might all be included under Ward's two classes.

Freeman (1), in essence, makes the same two main classes as does Ward. He terms them (a) organic diseases, and (b) inorganic diseases. The organic diseases are those due to the attacks of living organisms, and the inorganic diseases are those due to the non-living environment.

In 1915 Melchers (3) suggested the following grouping of plant diseases: (a) non-parasitic diseases; (b) diseases of unknown origin; and (c) parasitic diseases. It is evident that Melchers' classes (a) and (c) correspond to Ward's classes (a) and (b), respectively, while his class (b) constitutes a simple and convenient pigeon-hole in which to stow away temporarily those vexatious diseases of unknown cause which are discovered from time to time. It is strange that none of the earlier botanists thought of inserting such an item in their systems of classification. Perhaps their optimism concerning their own powers of discrimination prevented their seeing any necessity for such a class.

As our knowledge of the cause of plant diseases becomes more exact and as the types or phases of plant maladies which are conceded to come within the sphere of the plant pathologist become more clearly defined, it is possible to make a more exact classification of plant diseases. For example, neither Ward's nor Freeman's classification would exclude types of injury caused by insects which now by common consent are included within the field of the entomologist. When all the types of injury to plants which have gradually come to be included within the fields of entomology, bacteriology, or zoölogy have been excluded from plant pathology, and only those phases which have come to be regarded as exclusively within the field of the plant pathologist are considered, then it becomes possible to make a classificatory list which will include only the kinds of plant diseases strictly within the field of plant pathology and exclude all other types of injury. The following outline is the result of an attempt to make such a classification of plant diseases and is the scheme of classification which will be followed in Part II of this text.

#### I. Parasitic diseases.

##### A. Caused by parasitic plants.

##### 1. Caused by slime molds.

2. Caused by bacteria.
  3. Caused by fungi.
  4. Caused by algæ.
  5. Caused by parasitic seed plants.
- B. Caused by parasitic animals.
1. Caused by nematodes.
- II. Virus diseases (infectious chloroses).
- III. Non-parasitic diseases (so-called "physiological" diseases).
- IV. Diseases of unknown origin.

This outline needs a word of explanation. Perhaps the most questionable point centers around the place of the virus diseases in this scheme. Not so long ago the writer would have placed this group of diseases under point IV, diseases of unknown origin. At present, however, we know a good many things about the nature of these troubles even though we do not know the exact cause. We know so much about them, in fact, that there might be good reason for including them as a sub-group under parasitic diseases. But since no parasite has been positively identified as the causal agent, it has seemed best to place the virus diseases tentatively in a class coördinate with parasitic and non-parasitic diseases. If a causal parasite is finally demonstrated for this group of diseases, then of course they will have to be transferred to their proper position under parasitic diseases.

**Plant diseases classified on basis of symptoms.** — The fact that a great many of the early botanists were physicians who studied botany as an avocation accounts for most of the terminology formerly used in describing the symptoms of plant diseases. In 1773 Zallinger (9), in "*De Morbis Plantarum*," suggested the following classes of plant diseases based upon the symptoms: (a) phlegmasiae, or inflammatory diseases; (b) paralysis, or debility; (c) discharges or draining; (d) cachexia, or bad constitution; and (e) chief defects of different organs. This list of symptoms certainly sounds more like animal pathology than plant pathology, but even today the plant pathologist still uses many terms, in exact or modified form, that are used by the animal pathologist. Examples of present-day terms applied to plant diseases but borrowed from the medical profession are: canker, oedema, necrosis, embolism, and anthracnose. In some cases these terms have been modified but nevertheless in the original they were borrowed from human pathology. While it is manifestly impossible to make exact comparisons between the symptoms of disease exhibited by members of the animal kingdom and those observed in diseased plants, yet there are many resemblances,

fancied or real, which have led to the adoption, by the botanist or plant pathologist, of many of the terms used in the medical sciences.

At the present time probably the simplest and most convenient system of classifying plant diseases on the basis of symptoms is to use the three types of symptoms, discussed above under symptoms and signs of disease, as the basis for the three classes of diseases, namely the necrotic, the hyperplastic and the hypoplastic diseases. The three main classes of diseases, then, on basis of symptoms, may be described as follows: (a) diseases resulting in the death of the affected tissues, whether these tissues represent the whole plant or only a part of it; (b) diseases resulting in the slowing down or stunting of the growth of the plant or plant parts, and (c) diseases resulting in the overgrowth or overdevelopment of the plants or organs affected.

**Classification of diseases on basis of hosts.** — The classification of plant diseases on the basis of hosts is simply a matter of convenience. There is no fundamental principle involved in this system of classification. In many colleges and schools of agriculture it is customary to offer courses on the diseases of particular crops or groups of crops for the convenience of students interested in specialized fields of agriculture. For example, a course might be given for students specializing in vegetable gardening. Such a course might be entitled "Vegetable Diseases," or "Diseases of Vegetables." Likewise another course designed especially for students of pomology might be labeled "Fruit Diseases." A still more specialized course might be entitled "Diseases of Cane Fruits," or "Potato Diseases."

Such courses based on a host classification of plant diseases should as a rule follow a general course in plant pathology which should be prerequisite for the pursuit of the specialized course. At least, some knowledge of the classification of diseases both on the basis of cause and on the basis of symptoms should be acquired, if not in a general beginning course, then as a part of the special course. The most fundamental principle in the science of plant pathology is certainly the fact that the cause of a disease must be thoroughly understood before effective control measures can be applied intelligently. Any control measures not based on accurate knowledge of the etiology of the disease must necessarily be empirical and consequently many times more or less ineffective. Therefore, the causes of disease in plants should be accurately classified and each type of causal agent thoroughly understood. A knowledge of symptomatology is scarcely less desirable. A study of all the effects of the disease upon the host should be made, including the pathological anatomy or histology of the diseased parts. The accurate classification of the disease on the basis of symptoms is necessary for accurate diag-

nosis. Thus from the standpoint of acquiring the fundamentals of plant pathology, the classification of plant diseases as to cause and symptoms is of far greater value than a host classification alone.

### REVIEW QUESTIONS

1. Study the various definitions of plant disease given and decide upon the relative merits of each definition. Give reasons for your decisions.
2. What are the three general types of symptoms of disease in plants? Give examples of each.
3. What distinction is sometimes made between "symptoms" and "signs" of disease?
4. Name three different bases on which plant diseases may be classified. Discuss the merits of each basis of classification.
5. Give some historical examples of different systems of classification. Indicate what basis was used in each case.
6. Memorize the outline of classification to be used in Part II of this text.

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## CHAPTER IV

### METHODS OF INVESTIGATING PLANT DISEASES

Before proceeding very far in the study of plant pathology it is desirable that we gain some insight into the methods of research employed by workers in this science. The procedure will vary somewhat with the type of disease to be studied. For the purposes of this discussion we will consider the three chief classes of plant diseases, namely, the parasitic diseases, the virus diseases, and those of non-parasitic origin. Since the virus diseases resemble the parasitic diseases in some respects, the methods used in experimentation upon them will necessarily resemble in some ways the methods of studying the parasitic diseases; yet there are some differences between the two types of diseases which are reflected in the research methods applied to each. The non-parasitic diseases differ so radically from the other two that the methods of attacking the problems connected with them must necessarily differ in many respects from those employed in studying the parasitic diseases and those of virus origin.

#### Investigations of Parasitic Diseases

As a rule any very extensive and fundamental investigations of the parasitic plant diseases involve the growing of the organisms in pure culture and the making of artificial inoculations, or, in special cases, the latter operation only. Since by far the greater number of the parasitic plant diseases are caused either by fungi or by bacteria, this discussion will be confined to these two similar types of causal organisms. A great many fungi and bacteria can be grown in pure culture on artificial media of various kinds. A few common examples of such organisms are the brown-rot fungus, *Sclerotinia cinerea*; the peach-scab organism, *Cladosporium carpophilum*; the fire-blight bacterium, *Bacillus amylovorus*; the crown-gall organism, *Bacterium tumefaciens*; the apple-blotch fungus, *Phyllosticta solitaria*; and the potato Rhizoctonia fungus, *Cor-ticium vagum*. Such organisms are facultative. They are capable of living as active parasites on their hosts for a time and then changing their habits to the extent of being able to live as saprophytes on dead organic matter. Many organisms do this habitually in nature, living for a time as parasites, finally killing the host, or a part of it, and then surviving for a longer or shorter time as saprophytes in the tissues they have killed. The brown-rot fungus is a good example of this habit.

After it destroys a fruit by rotting it, the mycelium lives for a year or more in the mummified fruit, completing its life history as a saprophyte. As a rule organisms which are able to survive saprophytically in nature can be easily cultured on artificial media. On the other hand, certain kinds of fungi have thus far not been successfully grown in test-tubes or on a medium other than the living host. Examples of such obligate parasites are the rusts and the powdery mildews. (See Chapter IV.)

**Culture media and apparatus.** — In order that many of these organisms may be grown artificially for the purposes of experimentation, plant pathologists and bacteriologists have developed an extensive technique. In the first place it was necessary to discover the kind of medium on which the different organisms would grow best. In the next place a considerable amount of apparatus is required for preparing the media and for using them for growing the organisms. There are two general classes of media, namely, liquid and solid. Of course there are many variations and modifications of each class required for special cases but all of these details cannot be discussed here. Only a brief description of some of the more commonly used media and the methods of preparing them will be given in the following pages.

*Liquid media.* — These are more often used in the culture of bacteria than of fungi although they are sometimes used in culturing fungi for certain purposes. On the other hand liquid media are not used invariably in the culture of bacteria for many of these are often grown on solid media. Beef extract or bouillon is the chief liquid medium used for culturing bacteria. Milk also is sometimes useful in the artificial culture of some bacteria. Synthetic liquid media composed of various salts and other nutritive substances in proper proportions are necessary in certain physiological studies of pathogenic organisms. Synthetic media may also be solidified with agar for use in other cases. Many different formulae for synthetic culture media have been used by different workers. Some of the media of this kind are Naegeli's nutrient solution, Mayer's culture fluid, Uschinsky's solution, and Fermi's culture fluid. Many others have been used. As an example of the nature of one of these synthetic media the formula used by Mayer is given below:

Magnesium sulfate.....	10.0 gm.
Ammonium nitrate.....	15.0 "
Tri-basic calcium phosphate.....	.1 "
Potassium phosphate.....	10.0 "
Distilled water.....	1000.0 "
Dissolve cold and add sugar.	

For additional formulae for various kinds of culture media see *Bacteria in Relation to Plant Diseases*, Vol. I, pp. 195-201, by E. F. Smith.

*Solid media.* — In making cultures of fungi solid media are most commonly used. They are of two general types, namely, agar and pieces of various plants. Agar is a substance resembling gelatin and is manufactured from certain species of seaweeds. The agar alone has no great nutritional value but is simply used to solidify the nutrient medium

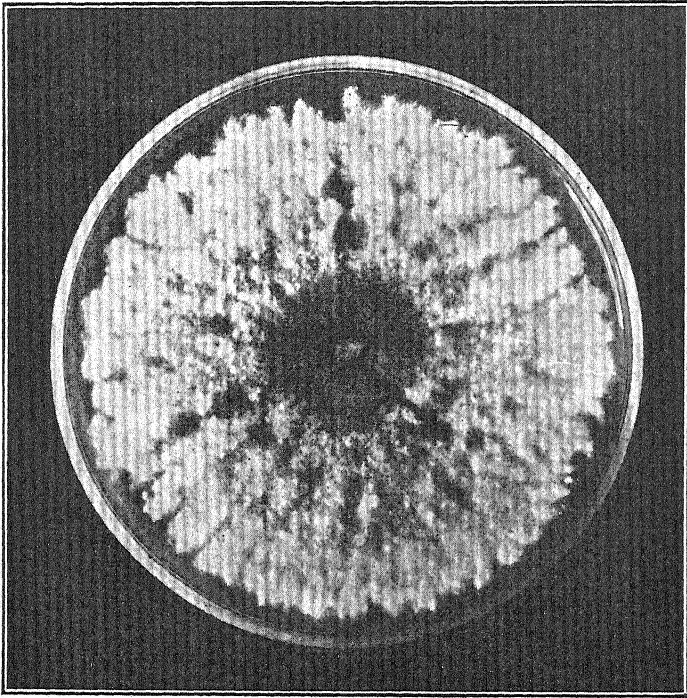


FIG. 1. — Pure culture of the clover stem-rot fungus (*Sclerotinia trifoliorum*) in a Petri dish on nutrient agar. (Photograph by O. H. Elmer, Ore. Agr. Exp. Sta.)

which usually consists of some plant decoction suitable for the growth of the fungus. In making up an agar medium it is customary to add a plant decoction of the desired kind to the melted agar and let the mixture solidify into a jelly-like mass. There are many different plant decoctions which have been used for this purpose but plant pathologists have come to rely upon certain standard formulae for ordinary work with the average run of fungi, and resort to special formulae only for particular kinds of work in which the standard formulae will not answer the purposes of the investigation.

A stock formula which works well with many common fungi is known as potato-glucose agar. It is sometimes made according to the following formula: 500 grams potatoes; 50 grams glucose; 50 grams agar; and 2500 cc. of water. The potatoes are cooked in enough water so that when done and the water is strained off there will be 500 cc. of it. The glucose is dissolved in 500 cc. of water. The agar is melted in 1500 cc. of water. When all three ingredients are ready they are mixed and sterilized in an autoclave for 15 minutes at 15 pounds pressure.

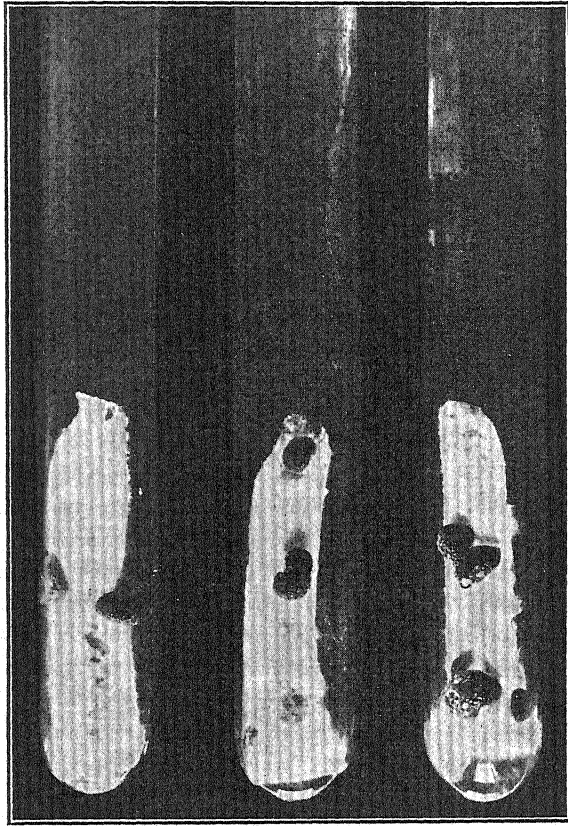


FIG. 2. — Cultures of the clover stem-rot fungus on sterile carrot plugs in test-tubes. The pieces of carrot are covered with a white mycelial growth and several black sclerotia have developed. (Photograph by O. H. Elmer, Ore. Agr. Exp. Sta.)

Then while hot the mixture is filtered through cheese cloth and cotton into test-tubes, about 10 cc. being allowed to each tube. The tubes are then plugged with cotton stoppers and sterilized again for 15 minutes.

Finally the tubes are placed in a slanting position until the agar cools and hardens.

Pieces of plants are frequently used without agar and without the addition of any other nutritive substance. For example, potatoes or carrots (Fig. 2) may be cut into suitable pieces, placed in test-tubes, plugged and sterilized in the usual way. Substances such as rice and corn-meal are sometimes used. Herbaceous stems may be used in the same way. For culturing some of the wilt-producing organisms such as *Fusarium* and *Verticillium*, sweet-clover stems are very satisfactory. The stems are simply cut into pieces 2 or 3 inches long and the pieces are placed in the test-tubes. A little water is added to each tube to supply moisture, and the tubes are plugged and sterilized in the usual manner.

*Apparatus.* — The tools and apparatus necessary in making cultures and inoculations fall into three classes: (a) the appliances used in cooking and sterilizing the media; (b) the containers for storing the media after it is sterilized and during the time the cultures are growing; and (c) the instruments used in making the isolations, transfers and inoculations. Gas burners, electric hot plates or steam from the heating plant or other source may be utilized to furnish the necessary heat for cooking and sterilizing the media. Granite-ware or aluminum cookers and kettles should be available for use in preparing the materials. Every well-equipped laboratory is supplied with autoclaves or other types of steam sterilizers. For the storage and utilization of the different media after they are prepared, flasks, test tubes and Petri dishes are needed. Cotton batting for plugging the tubes and flasks, and cheese cloth, cotton and funnels for filtering the hot agar are necessary. Certain tools are also necessary in making isolations and inoculations. Scalpels, dissecting needles, inoculating needles and platinum loops are the instruments most commonly needed in this work. Aluminum beakers also are useful at times. For further details on culture media and apparatus see references 1, 5 and 6 at the end of this chapter.

*Isolations and inoculations.* — There are several different methods of isolating an organism in order that it may be grown in pure culture. The method used depends upon the nature of the organism, the stage present on the diseased specimen and the nature of the substratum. If the organism to be cultured is a fungus it may be present only in the mycelial stage. In this case the method used in isolating it may be different from the method which would be used if the fungus were sporulating freely. In the former case the tissue-culture method will be used while in the latter case spore cultures may be made. If the disease is of bacterial origin the methods of bacteriology will be used in culturing

the organism. The technical procedure in making spore cultures and in culturing bacteria is practically identical.

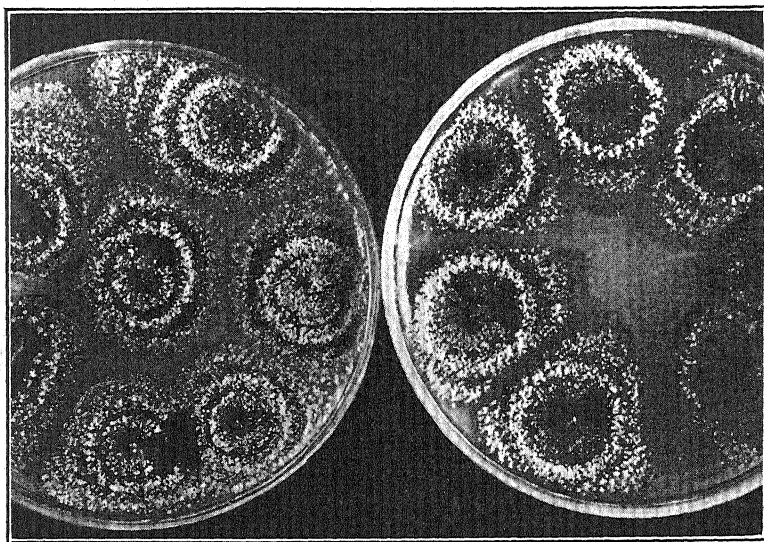


FIG. 3. — Pure culture of the brown-rot fungus in a Petri dish, on nutrient agar, showing separate colonies each originating from a separate inoculation. (Photograph by G. B. Posey, Ore. Agr. Exp. Sta.)

*Tissue cultures.* — This method may be used whether or not the fungus is producing spores, but in case no spores are present it must be used. It consists essentially in removing bits of the diseased tissue from the host and depositing these fragments on the culture medium. In many cases, as, for example, rotted fruits, bark cankers and vascular diseases of potato tubers, the mycelium of the fungus is present in abundance in the diseased tissues, and when pieces of the affected tissue are transferred some of the mycelial threads are carried along and deposited on the medium where they continue to grow. After a few days the mycelium will spread out over the surface of the agar and small bits of the new growth may be transferred to other tubes or plates and thus as many pure cultures of the fungus as are desired may be secured. In this procedure it is essential to use every precaution against contaminating the culture with other organisms. Suppose it is desired to isolate the organism causing an apple fruit rot. The mycelium of the causal organism is present beneath the surface of the fruit in the rotted tissue. But there may be other organisms, bacteria or spores of other fungi, on the skin of the apple, although invisible. If the work is done



carelessly some of these stray organisms may be carried along with the desired one into the culture medium and thus contaminate the culture. In this way several organisms may be found growing in the culture tube instead of just the single one desired. To avoid this, great care must be taken. In the first place the work must be done in a room that is as nearly sterile as possible and all apparatus, table tops and tools must be sterilized. In the second place the surface of the diseased specimen should be sterilized before cutting into it. The following procedure carefully followed should give good results. Wash the surface of the diseased specimen with a disinfectant such as alcohol or corrosive sub-

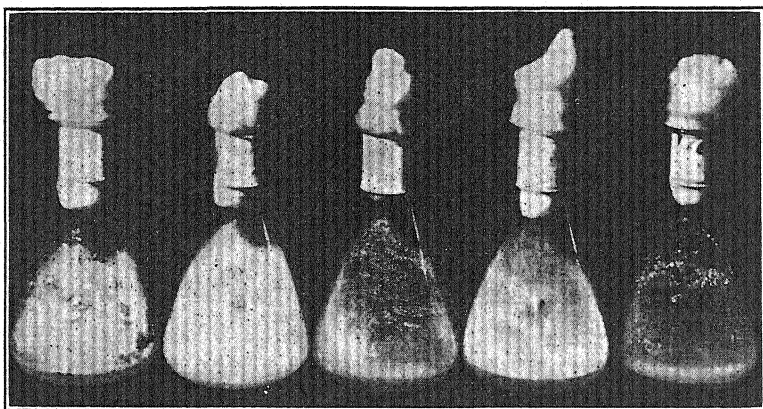


FIG. 4. — Cultures of the potato *Rhizoctonia* fungus in flasks on nutrient agar. (Photograph by J. H. Corsaut, Ore. Agr. Exp. Sta.)

limate, or hold it temporarily over a flame such as a gas burner or an alcohol lamp. The sterilizing process should be such as to kill any foreign organism on the surface without injuring the mycelium of the fungus imbedded in the tissues and which it is desired to culture. Then, by using flamed scalpels and needles, cut into the tissues and carefully transfer small fragments, not much larger than pinheads, to the culture medium. The plugs in the test-tubes should be flamed before being removed, and kept out only long enough to “plant” the bits of tissue on the surface of the agar. If Petri dishes are used, lift the lids carefully just sufficiently to permit of depositing the inoculum in the dish, then replace the lids quickly. The tissue-culture method is a very useful one and can be used successfully in a great many cases where the mycelium of the causal organism is growing in “pure culture” in the host tissue. Of course, if more than one organism is occupying the same region this method cannot be successfully used. It rarely happens, however, that “mixed cultures” occur in such diseases in nature.

*Spore cultures.* — In case the fungus is sporulating the spores may be used instead of the mycelium in making isolations and cultures of the organism. For making cultures of this sort the following apparatus and materials are required: test-tubes containing a few cubic centimeters of sterile water, sometimes termed "water-blanks"; other test-tubes containing agar; sterilized Petri dishes; a platinum needle formed into a loop; a beaker with cheese-cloth or cotton in the bottom of it for holding the tubes of agar; and a thermometer. The agar should be melted by heating the tubes in a beaker of water or in a steam sterilizer. The melted agar must be held at a temperature just above the point at which it solidifies, that is, at about 40° to 42° C. When everything is ready proceed in the manner described below.

Place some of the spores in a tube containing a small amount of the sterile water. Shake thoroughly so as to diffuse the spores as much as possible. Then insert the platinum loop into this tube and transfer a loop of the water containing suspended spores to a tube of the melted agar, which is being held just above the melting point. After making this transfer, shake the agar tube thoroughly and transfer a loop from it to a second agar tube. Shake and repeat the transfer to a third tube containing melted agar. Then pour the agar from tubes one, two and three respectively into three Petri dishes. Set the dishes away to cool and let stand until the spores germinate. This method of making a series of three cultures, each succeeding one of which is more dilute than the preceding, should give at least one plate in which the fungus colonies resulting from spore germination should be sufficiently isolated from neighboring colonies to permit of easy transfer of the fungus to new tubes in pure culture. The plate can be examined under the microscope to determine if any particular colony arises from a single spore. Bits of agar containing single-spore colonies can then be cut out and transferred

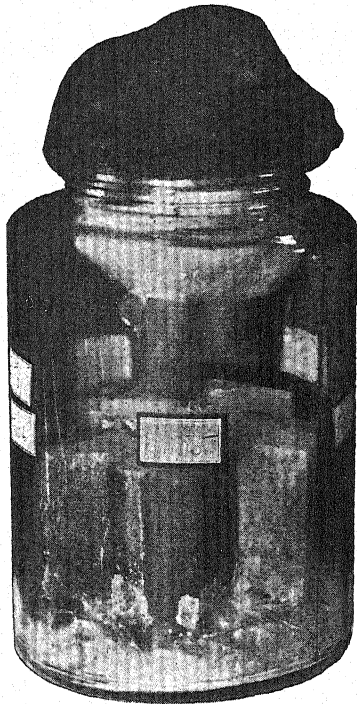


FIG. 5. — Method of growing wood-rotting fungi on sterile blocks of wood in glass jars. (After Zeller, Ann. Mo. Bot. Gard. 4: 93-164.)

to permit of easy transfer of the fungus to new tubes in pure culture. The plate can be examined under the microscope to determine if any particular colony arises from a single spore. Bits of agar containing single-spore colonies can then be cut out and transferred



to new tubes and thus pure cultures containing single-spore strains of the fungus can be secured.

*Bacterial cultures.* — In isolating a bacterial organism from a diseased plant, first sterilize the surface as described above, then crush a bit of the infected tissue in a tube of sterile water by means of a sterile glass rod or other suitable instrument in order to release large numbers of

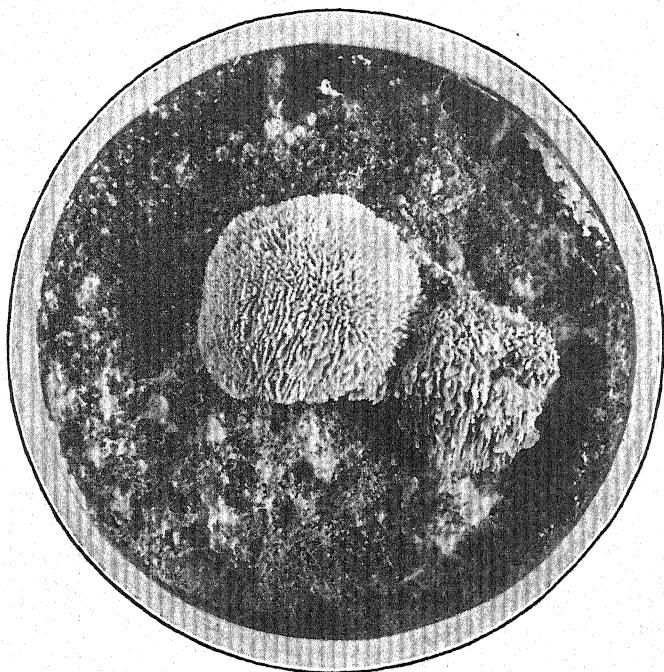


FIG. 6. — A wood-rotting fungus (*Lenzites sepiaria*) grown and fruiting on a sawdust medium in a glass flask. The top of the flask has been broken away so that the fungus could be more readily photographed. (After Zeller, Ann. Mo. Bot. Gard. 3 : 439-512.)

the bacteria in the water. Then the further procedure is exactly as described above for spore isolations. If these cultures are made sufficiently dilute it will be possible to pick out colonies on the plates which have arisen from a single bacterium and subcultures can then be made from these colonies with assurance of getting a pure culture of the organism.

*Inoculations.* — After the organism has been secured in culture, it is usually easy to make inoculations into healthy plants. If the organism is one that cannot enter the host through the unbroken epidermis, the

inoculation is usually made by cutting through the epidermis with a sterile scalpel or puncturing it with a needle. Then either spores or bits of the mycelium are inserted into the opening and some device is used to prevent too rapid drying out of the wound. On the other hand, if the organism is able to enter in the absence of any wound in the surface of the host, inoculation may be accomplished by simply placing some spores on the surface of the host by means of such instrument as

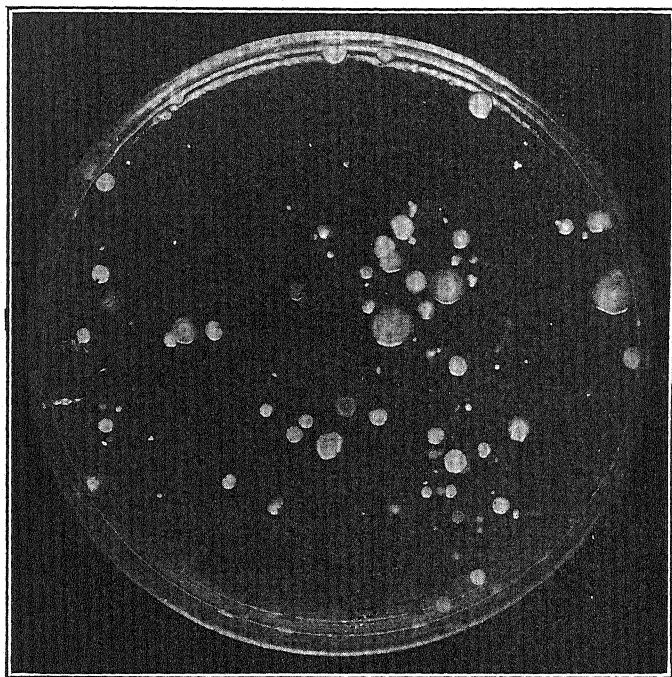


FIG. 7. — Culture of bacteria in a Petri dish on nutrient agar.

a camel's hair brush, or they may be sprayed on with an atomizer. In this type of inoculation it is usually necessary to spray the inoculated surface thoroughly with water and retain a high degree of humidity by keeping the inoculated plant in a moist chamber or by some other effective device. The rusts are good examples of fungi with which the latter type of inoculation is effective. With the rusts no cultures on artificial media are possible or necessary. Fresh spores can simply be taken direct from one plant and placed upon another as described above. Inoculations with bacteria may be made in the same manner as with fungus spores. In some cases wounds are necessary while in other cases simply spraying the bacteria on the surface is sufficient.

**Some uses of pure cultures and artificial inoculations.** — There are many different reasons for making inoculations. Some of the purposes for which the plant pathologist may make artificial infections are to determine: (a) the cause of the disease; (b) conditions for growth of the organism; (c) susceptibility of varieties; (d) life-history relationships; (e) biological races; (f) conditions for development of different stages; (g) methods of control.

(a) *To determine the causal relation of a particular organism.* — An example will serve to illustrate this point. The causal organism of apple anthracnose (*Neofabraea malicorticis*) can easily be isolated by the tissue-culture method previously described, grown in pure culture and re-inoculated into a healthy tree by cutting the bark and inserting a bit of the culture, with the result that in due time a typical canker will appear on the tree. In this connection the plant pathologist should keep constantly in mind that the rules of proof, which Koch formulated for use in studying disease-producing bacteria, and which are known to the bacteriologist as "Koch's Postulates," are just as applicable to the study of fungous and bacterial diseases of plants as to the work of the bacteriologist and physician. As applied to plant pathology these rules may be stated as follows:

(a) An organism which is always found associated with a particular diseased condition of a plant, (b) should be isolated and grown in pure culture, then (c) inoculated into a healthy plant of the same kind and result in the characteristic disease, and finally (d) the organism should be reisolated from the second plant and compared with the first culture. Both the diseased condition induced by inoculation, and the organism recovered from the inoculated plant should correspond to the original diseased condition, and to the first organism isolated, respectively. (See references 1 and 3.)

It is only when all of these steps have been taken that one is justified in concluding that any particular organism is the cause of a certain disease.

There are some exceptions in which these rules of proof cannot be rigidly followed. It is only when working with an organism that is capable of spending at least a part of its life cycle on artificial media that Koch's Postulates can be carried out in full. Certain pathogenic organisms are obligate parasites and therefore cannot be grown in artificial cultures. Consequently, in working with a disease caused by such an organism, the steps pertaining to artificial cultures of the organism must be omitted.

Reference to obligate parasitism leads to the problem of accurately classifying plant disease organisms as *parasites* or as *saprophytes*. Many

of them are not entirely one or the other. Some organisms which are parasitic in one stage are saprophytic in another stage, and *vice versa*. This has led to their tentative classification under four heads, namely: (a) obligate parasites; (b) facultative parasites; (c) obligate saprophytes; (d) facultative saprophytes. In such a system of classification the obligate parasites are those which cannot live on any substratum other than the living host, as illustrated by the rusts in their familiar phases. A facultative parasite is one which, while ordinarily a saprophyte, can, under the proper conditions, become parasitic, as, for example, some species of *Fusarium* and other fungi which are responsible for "sick soil" conditions. An obligate saprophyte is an organism that always lives on dead organic matter and cannot become parasitic under any conditions. Certain molds and bacteria of decay may be classed here. A facultative saprophyte is one that is usually parasitic but upon occasion may become a saprophyte. This classification is only tentative and there must necessarily be more or less overlapping of the various classes because among plant disease organisms there are many degrees or gradations between those that are obligate parasites and those that are obligate saprophytes. In many fungi the perfect stage is saprophytic while the conidial stage is parasitic, as illustrated by the apple-scab fungus, *Venturia inaequalis*, the brown-rot fungus, *Sclerotinia* spp., and the cherry leaf-spot fungus, *Coccomyces hiemalis*. In the case of the corn-smut fungus, *Ustilago zeae*, the sporidia may develop indefinitely as saprophytes, whereas the fungus is parasitic throughout its life on the host. Stevens and Young (10) suggest the terms, *totoparasite* to designate a fungus that is parasitic in both its perfect and imperfect stages, and *tropoparasite* or cases in which the conidial stage is parasitic and the ascigerous stage saprophytic.

(b) *To determine the conditions under which an organism is most virulent.* — It is a well-established fact that the environment plays a very important part in determining the prevalence of any disease. The soil and climatic or weather conditions must be taken into consideration in any intensive study of parasitism. It is well known that acidity and alkalinity of the soil determine the ability of certain organisms to become serious pathogenes. The temperature and the moisture content of the soil as well as of the air are important factors, the fluctuations of which favor or inhibit, as the case may be, the development of disease organisms. Artificial inoculation experiments are of great aid in studying the influence of such conditions upon the activity of the organism. The influence of environment upon plant diseases will be discussed in more detail in Chapter V.

(c) *To determine the susceptibility of varieties, species, etc., to a specific*

*organism.* — It is often desirable to determine the range of pathogenicity of a particular organism. For example, it is desirable to know just what species of pine, gooseberries and currants are susceptible to the white pine blister-rust fungus, *Cronartium ribicola*. Rather than depend upon observations of natural infection, extending over a long period of time and a wide range of territory with no certainty as to the final conclusions, it is possible and desirable to bring together in one place all known species of the above-mentioned hosts and, under the most favorable conditions, make artificial inoculations and thus determine in a relatively short time exactly which species are susceptible to attack and to what extent. Of course the conditions for infection in such an experimental plot must be ideal in order to get the most reliable results.

(d) *To determine the life history relationships of the different stages of an organism.* — Many fungi have two or more stages in their life cycle. These stages usually do not occur simultaneously but follow each other in rotation. In many cases the different stages may occur on different parts of the same host or even on alternating hosts. The brown-rot fungus has two spore forms, the conidial (*Monilia*) stage and the perfect or ascospore stage. The conidial stage appears on recently rotted fruits, on blighted blossoms and spurs and on branch cankers. The ascospore stage develops from the sclerotia of old mummified fruits that have lain on the ground for one or more seasons. Before these two spore forms could be definitely ascribed to the same species beyond the shadow of a doubt, it was necessary to grow the fungus in pure culture and by inoculating the host to produce the spore stages again. For example, a pure culture could be grown from a single ascospore. Upon inoculating a fruit from this culture and allowing the fruit to rot under proper conditions a crop of conidiospores would be produced. Then if the rotted fruit were planted out on the ground and allowed to remain under favorable conditions for the requisite period of time the perfect or ascospore stage would appear. This same procedure might be carried out by starting with a single conidiospore and the same results obtained. After all these steps have been successfully carried out there can no longer be any doubt as to the relationship of the two stages.

Another good illustration of this point is the classical example of De Bary's work with the black stem rust of wheat which has several spore stages on two different hosts. While this rust fungus cannot be grown in pure culture in a test-tube as can the brown-rot fungus, yet it is possible to inoculate wheat plants with aeciospores from the barberry and produce the urediniospores and teliospores on the wheat, or to inoculate barberry bushes with basidiospores from the telial stage and produce the aecial stage on the barberry.

(e) *To determine if an accepted species can be segregated into biologic forms.* — One of the best illustrations of the work that has been done on the so-called “physiological species,” or biologic races, strains, or forms, is that done by Stakman (7) and others (8, 9) on stem rust (*Puccinia graminis*). This species has been divided into three chief races, namely *Puccinia graminis tritici* on wheat, *Puccinia graminis secalis* on rye and *Puccinia graminis avenae* on oats. The form *tritici* has again been subdivided into several other forms. All of this segregation has been accomplished by careful inoculation work. It has been found that some species or varieties of grains are susceptible to certain races of the fungus and resistant or immune to other forms. Stakman and his co-workers have selected a dozen varieties of wheat which they have determined to be most useful in differentiating the known biologic forms of *Puccinia graminis tritici* and have designated these as “differential hosts.”

(f) *To test certain methods of control.* — In some cases experimental work on plant-disease control can be much more readily and accurately carried out if the host can be artificially inoculated with the disease organism. Reimer (4) performed extensive tests with wound disinfectants for fire-blight by first inoculating a freshly made wound with a pure culture of the fire-blight organism, *Bacillus amylovorus*, and immediately applying the disinfectant to the surface of the wound. A type of inoculation is often used in experimental seed treatment for the control of wheat smut. A quantity of wheat is thoroughly dusted with smut spores and then divided into two parts. One part is treated with the fungicide to be tested while the other portion is left untreated and planted as a check.

(g) *To determine the conditions under which the different stages of a fungus will develop.* — In some cases the perfect stage of a fungus is rarely found while the conidial stage may appear in abundance every year. In such cases it would seem that the stage which occurs only rarely is very sensitive to environmental or other conditions and shows up only under just the right combination of circumstances. It is often desirable to determine what these favorable or unfavorable conditions are and the surest way is by means of artificial culture work. For example, Leonian (2) found that he could induce or prevent the formation of both pycnidia and perithecia in *Valsa leucostoma* by varying the ingredients in the culture medium on which the fungus was grown.

### Investigation of Virus Diseases

While the virus diseases resemble the parasitic diseases in some respects, they differ from them sufficiently to make necessary some modi-

fications in the research methods used in studying them. The chief difference, however, lies in the fact that as yet no method has been devised for growing the causal agent of the virus diseases in pure culture as most fungi and bacteria can be grown. The fact that the causal agent is found in the sap of the plant makes it possible to carry on inoculation work with these diseases in much the same manner as with the fungous and bacterial diseases even though the causal agent cannot be isolated and grown in test-tubes. For details of making inoculations with the virus diseases see the special discussion of these diseases in Chapter XXIV.

In view of the fact that the different virus diseases cannot be identified by studying the characteristics of the causal agent under the microscope

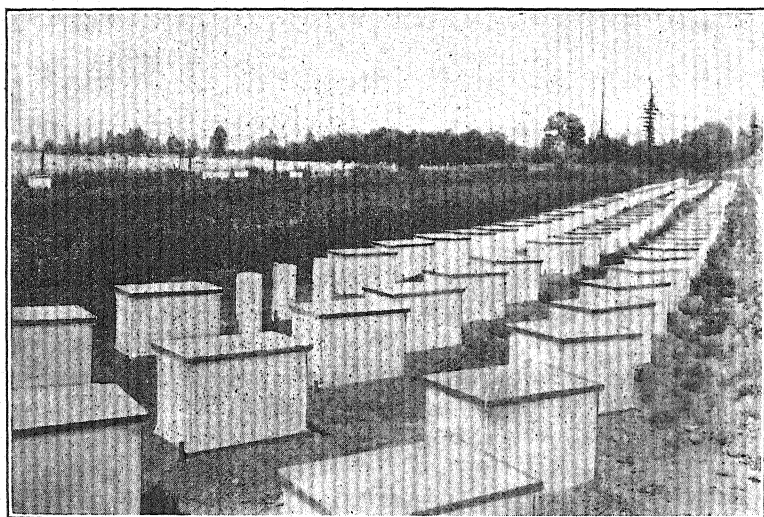


FIG. 8. — A layout of insect cages used in the investigation of potato virus diseases. (Photograph by McKay, Ore. Agr. Exp. Sta.)

and in pure culture as is the case with the fungous and bacterial diseases, it became necessary to work out new methods which are better adapted to the study of this type of disorder. The methods consist of two chief items, namely, the making of inoculations with juice from diseased plants, and the observance of the symptoms produced. The only way of judging as to the performance of a virus when inoculated into a plant is to observe the symptoms produced. Some of the reasons for making inoculations in studying the virus diseases are similar to the reasons given under parasitic diseases. As applied to the virus diseases these reasons are as follows: (a) to determine if a certain type of symptom is



due to a virus; (b) to segregate different types of virus diseases; (c) to determine the host range of a specific virus; (d) to determine the conditions under which the characteristic symptoms are evident and the conditions under which they are masked; (e) to determine the different methods by which virus diseases may be transmitted.

Another important procedure which has recently come into use in dealing with the virus diseases of potatoes is the tuber indexing system developed as an aid in the selection of virus-free seed. (See Chapter XXIV for a further discussion of this practice.)

Another very important feature of investigational work on virus diseases is the necessity for using insect cages in all outdoor inoculation work (Fig. 8). The fact that many of the virus diseases are known to be transmitted by insects makes this necessary. Thus if the experimental plants were not protected by insect-proof cages against chance infection from this source, the results of inoculation experiments would be largely invalidated.

### Investigation of Non-parasitic Diseases

Since this type of disease is non-infectious, neither cultures nor inoculations can be used in investigating the non-parasitic diseases. They are due chiefly to environmental factors, and this fact determines the methods which must be used in studying them. Research work on this type of disease, therefore, must necessarily consist largely of a study of the effects of environmental factors and how to overcome them. These factors may be roughly divided into two classes, namely, (a) the natural factors such as soil, climate and weather; and (b) the artificial factors such as cultural practices and storage conditions. Under the first group of factors it is necessary to study the effect of differences in temperature and moisture content of both the soil and the air. Under the second group are included such items as methods of cultivating; cover crops; pruning; time of harvesting with reference to maturity; time of storing; and the temperature, humidity and ventilation of storage quarters and transportation carriers.

**Records.** — In all investigations of plant diseases of whatever nature, it is necessary to keep accurate records of everything that is done and of all results obtained. In the case of the parasitic diseases, the items to be recorded include descriptions of the symptoms on diseased specimens; accessioning specimens for future reference; source of the material and date collected; date of culturing and of any transfers; kind of culture medium used; conditions under which cultures are grown; growth characters, and rate of growth; inoculations made, how, when



and under what conditions; results of inoculations; and records of weather conditions such as temperature and humidity. In fact it would be impossible to enumerate here all the items that should be included in the records. Every investigational problem will call for its own system of records to fit the peculiar circumstances. The point to be emphasized is that accurate and complete records should be kept of everything which may be of any value in this or future investigations. An important and valuable type of record in many cases consists of photographs and drawings. They should be used wherever circumstances dictate their usefulness. In the investigation of the virus and non-parasitic diseases similar full and complete records should be taken. The particular items to be recorded will vary in any case with the nature of the disease and the purpose of the investigation. The object in view and the circumstances under which the investigation is conducted must dictate the nature of the records kept.

#### REVIEW QUESTIONS

1. What practice is generally involved in any extensive investigations of the parasitic diseases?
2. What equipment and materials are necessary for growing pathogenic organisms in artificial cultures?
3. Describe the preparation of some of the common media used in this work.
4. Describe the process of isolating and culturing a fungus, both tissue cultures and spore cultures. Describe the process of isolating a bacterial organism.
5. How are inoculations made?
6. In case of a fungus, such as the rusts, which will not grow in a test-tube on artificial media, what procedure must be followed?
7. Mention the different uses to which pure culture and inoculation work may be put.
8. State Koch's postulates.
9. Define the following: obligate parasite; facultative parasite; obligate saprophyte; facultative saprophyte.
10. Compare and contrast the methods of investigating virus diseases with those used in investigating fungous and bacterial diseases.
11. Discuss methods of investigating non-parasitic diseases.
12. Discuss the necessity for and the manner of keeping records in this type of work.

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## CHAPTER V

### PLANT DISEASES AS RELATED TO ENVIRONMENT

One of the most important of the botanical sciences is ecology, the science which deals with the relation of plants to their environment. In a broad general sense the health and vigor of all plants depend upon the various factors of the immediate environment. Familiar examples of the influence of environment upon the common crops of field and garden may be cited here. That crops do not develop normally during a drouth or that corn, for example, will "drown" out in low, wet ground is universal knowledge, particularly among the agricultural class. The agriculturist knows that his crops will not yield as abundantly if he allows the weeds to grow unhindered in his fields as they would were the weeds relentlessly eradicated. He also knows that most agricultural crop plants do not grow as vigorously in a shaded situation as in one fully exposed to the sun, or in a thin clay soil as in a rich loam soil. These examples are mentioned here to show that almost any layman is familiar with many of the phenomena which illustrate the effect of ecological factors upon plant growth. However, in this discussion we shall deal more particularly with the relation of environment to the more specific ailments of plants which are generally considered to be within the field of plant pathology as distinguished from other plant sciences. All plant diseases, whether parasitic, non-parasitic or of virus origin, are profoundly influenced by environmental factors. The non-parasitic diseases are in large part, if not entirely, caused by the various factors of the environment. The occurrence of epidemics of the parasitic diseases is largely determined by environmental factors, and among the virus diseases the symptomatic expression of the disease depends to a great extent upon the climatic or weather conditions under which the host plant is growing. In the following pages the various environmental factors will be analyzed and illustrations of their relation to plant diseases cited. Particular attention will be given to the parasitic diseases in this connection, although the virus diseases and especially the non-parasitic diseases will not be neglected.

**Factors of the environment.** — The environment of plants is composed of two chief factors, namely, (A) the soil, and (B) meteorological conditions. The soil factor in turn consists of three elements: (a) the

physical condition of the soil; (b) its chemical composition; and (c) its biological condition, that is, its floral and faunal content. The chief elements of the climatic or weather factor are two, namely, (a) temperature, and (b) moisture. In so far as the last two items are concerned, no sharp line can be drawn between the soil and the meteorological factors, since under natural conditions both the temperature and the moisture content of the soil depend upon climatic and weather conditions.

**The soil.** — In discussing the soil as an environmental factor influencing the occurrence and development of plant diseases it should be remembered that there is such a close relationship existing between the soil factor and the meteorological factor that, in many cases, at least, neither one alone is responsible for the existence of any given condition with reference to disease occurrence. When considering any plant disease in relation to the environmental influences which are responsible for its occurrence we must not overlook the fact that more than one factor may be active at any given time, and we should study it not only in relation to the physical, the chemical or the biological aspects of the soil environment, but also in relation to the temperature and moisture factors.

*Physical condition.* — The physical make-up of the soil has a decided influence upon the growth of plants. Important items in the physical nature of the soil are the size and shape of the constituent particles and the looseness or compactness of the soil. These conditions in turn determine the water-holding capacity and the degree of aëration of the soil. While these factors have their influence upon plant growth in general, we are here particularly interested in the relation which these varying physical conditions bear to the occurrence of specific plant diseases. In so far as the soil texture has a great deal to do with its water-holding capacity this factor is related to the occurrence of parasitic diseases, since many of them are sensitive to the amount of moisture in the soil. This point, however, will be discussed in more detail under climatic and weather conditions as related to plant diseases. It is perhaps in connection with certain of the non-parasitic diseases that the nature of the soil from a physical standpoint bears a more direct relation. Several diseases of this nature, such as bitter-pit of apples, internal brown-spot of potato, and point-rot of tomato are possibly induced or at least intensified by soil conditions which are not conducive to a uniform water supply throughout the growing season.

*Chemical composition.* — The chemical reaction of the soil has an influence upon many plant diseases. There are striking illustrations of this relation to be found in the behavior of certain of the parasitic diseases. This applies particularly to soil-borne diseases. A very

familiar example of a disease which is influenced by the hydrogen-ion concentration of the soil is the common scab of potato. Workers agree that this disease is apt to be more severe in an alkaline soil. In experimental work the correction of this alkalinity has lessened the disease. In contrast with this case it has been shown that an acid soil is favorable for the development of the cabbage club-root organism, *Plasmiodiophora brassicae*. Neutralizing the soil acidity brings about a condition less favorable for the growth of the causal organism, hence a commonly recommended control measure for this disease is the application of lime to the soil. A striking correlation is also shown between the prevalence of the Fusarium wilt of tomato seedlings and the soil reaction. Sherwood (25) conducted experiments with this disease in which he used a series of soils, ranging from strongly acid to alkaline, such that the reaction of each soil in the series was accurately known. He found that the highest percentage of wilt occurred in the most acid soils of the series and that the amount of wilt decreased as the hydrogen-ion concentration of the soils decreased, within certain limits. Johnson (11) found that the growth of the tobacco root-rot fungus, *Thielavia basicola*, is checked by a very high soil acidity.

*Biological condition.* — The flora and fauna of the soil, especially the microscopic plant life, fungous and bacterial, are of the utmost importance to the plant pathologist. There are many different types of soil organisms, all of which affect plant growth either beneficially or harmfully. Of the former may be mentioned especially the bacterial organisms concerned in the nitrogen cycle so essential to plant nutrition. A deficiency in the supply of such organisms results in a lack of normal vigor and health in the crop grown on such soil. But the plant pathologist is more particularly concerned with the harmful microorganisms of the soil. Many of our most destructive plant diseases are caused by organisms which survive for a considerable time in the soil. Some soils become so thoroughly contaminated with the disease germs which attack certain crops that it is no longer possible to grow these crops profitably in such soils. Examples of diseases caused by microorganisms which are harbored in the soil are many of the Fusarium diseases such as flax wilt, the Fusarium wilt and rots of potatoes, onion smut, the corn root-, stalk-, and ear-rot, and the Rhizoctonia disease of potato and other plants. When one of these organisms becomes so abundant in a soil that its host crop can no longer be grown profitably, the condition is sometimes spoken of as "sick soil," and such terms as "flax-sick" soil or "wheat sick" soil are also used to designate the condition existing where a particular crop is affected in this manner. (For a more extended discussion of "sick soil" conditions see Chapter X.)

**Climate and weather.** — Discussions of plant diseases in ancient and medieval literature are full of allusions to the influence of the weather upon the various disorders to which plants are subject. Attention was called in Chapter I to certain superstitions that were prevalent among the ancients as to the part played by the weather, the planets and the gods in bringing about the diseases with which they had to contend in raising their crops. There was undoubtedly more truth in their beliefs concerning the influence of the weather than in any of the other evil influences to which they ascribed the diseases affecting their cultivated plants, but it is also true that they had no real conception of the exact manner in which climatic and weather factors do influence the inception and development of epidemics of plant diseases. Yet, in spite of the fact that their notions concerning the relation of weather to plant diseases were based more upon superstition than upon scientific facts, it still is significant that people in those days were thoroughly convinced that the weather, in some way, was responsible for the prevalence of many of the plant diseases with which they had to contend. These deductions on the part of ancient peoples were based almost entirely upon their observations that certain weather conditions were accompanied by outbreaks of disease. Present-day plant pathologists are equally certain that meteorological factors are closely related to the occurrence of plant diseases but modern plant pathology attempts to explain this relationship on a scientific basis.

The important items of the climate and weather factor are temperature and moisture. These have to be considered in connection with both the soil and the air. The temperature and humidity of the air bear a particularly close relationship to those parasitic diseases which are disseminated through the air and infect the aerial parts of plants. Air currents are also frequently of importance in the dissemination of this type of disease. With the soil-borne type of parasitic diseases the soil temperature and soil moisture are of much importance in determining to a very great extent the seriousness and even the existence of such diseases. The critical points in the life cycle of the organism at which temperature and moisture wield a determining influence are spore germination, infection, development of the disease, and the fructification of the organism. There are maximum, minimum and optimum degrees of temperature and percentages of moisture at which any of these processes in the life history of the parasite may occur. These factors of the environment also affect the vigor of the host, which in turn is reflected in its susceptibility or resistance to the disease.

There are many references in modern plant-disease literature calling attention in a general way to the effect of weather conditions upon the

✓ prevalence of specific plant diseases. The literature on peach leaf-curl, for example, contains numerous allusions to the influence of the early spring weather upon the occurrence of that disease. In 1900 Pierce (20) gave good reasons for suspecting that a cold, wet spring favored the development of the disease. He thought that such conditions were conducive to the growth of the leaf-curl fungus, *Exoascus deformans*, and also rendered the tissues of the young peach leaves more susceptible to invasion. Many general statements concerning the influence of weather conditions upon the prevalence of plant diseases may be found in various numbers of the Plant Disease Reporter issued by the Plant Disease Survey of the U. S. Department of Agriculture. In 1924 that publication (1) contained the following statement:

"The unusual weather conditions that have prevailed this season deserve especial mention in any report of the plant disease situation. In general it has been unusually cold in states east of the Rocky mountains and unusually wet for the most part east of the Mississippi river. On the other hand, west of the Rockies, drouth and hot weather have prevailed . . . . These unusual conditions, . . . especially the excess rainfall in the East, are bound to affect profoundly the plant diseases of the year. Already an epiphytotic of sycamore blight (*Gnomonia veneta*) has appeared and apple leafspot (*Physalospora*) is unusually abundant in some sections. On the other hand certain other diseases are being delayed in their appearance and may prove much less conspicuous than usual."

The same publication (Vol. IX, No. 3), issued July 15, 1925, reported many diseases, such as apple-scab and apple-blotch, to be rare in some states of the East and Mid-west on account of the extremely dry weather prevailing during the spring months of that year. It is a well-known fact that the brown-rot of stone fruits is more prevalent during periods of high humidity combined with warm temperatures. Epidemics of the black stem rust of wheat are closely related to periods of high temperature and abundant precipitation during the time when the wheat plant is rapidly approaching maturity.

While the above statements are more or less general there is not lacking an abundance of more specific evidence as to the effect of climatic and weather conditions upon plant diseases. During recent years a great deal of experimentation has been done on problems connected with the influence of soil and air temperature and moisture upon the growth of disease organisms and their ability to become virulent parasites. Jones (12), in 1917, expressed a conviction that plant pathologists will have to give more and more attention to the ecology of plant diseases. He called attention to a considerable amount of work that

had already been done on the effect of temperature and moisture on plant diseases and suggested that certain soil organisms such as the *Fusaria* lend themselves to studies of this kind because soil temperatures and moistures can be controlled under experimental conditions. Again, in 1924, the same writer (14) reiterated his plea for more intensive study by plant pathologists of the environmental factors in relation to plant diseases and urged that more attention be given to physiological studies in this connection and less to the mycological phases of plant-disease problems. A few examples of this type of work will serve to emphasize the importance of these ecological factors in the study of plant diseases. For the sake of simplifying the studies it is customary to study only one factor at a time, as, for example, soil temperature. In making a study of the effect of soil temperature it is necessary to hold other factors, as soil moisture, at a constant while varying the temperature factor. The reader is cautioned, however, against supposing that, in all cases, any one factor acts alone, although in some cases the evidence shows that a single environmental factor may determine the occurrence or non-occurrence of a particular disease in a given locality or during a certain season. In some instances it is evident that more than one factor, as, for example, both temperature and moisture, work together to determine the prevalence of a particular disease. This complicates the situation so that a complete investigation of this type of problem is not always as simple as some of the following experiments might seem to indicate.

*Effect of soil temperature.* — The investigation of soil temperatures in relation to plant diseases originated and developed largely at the University of Wisconsin. The equipment for carrying on such experiments consists essentially of water tanks, fitted out with automatic temperature controls, and in which soil containers can be placed so as to maintain any desired temperature in the soil (see reference 13). The experimental results cited below were obtained for the most part by the use of such equipment and methods.

Jones, McKinney and Fellows (15), working on the influence of soil temperature on the common scab of potato, reported that the scab organism is favored by a relatively high temperature (about 22° C.) while the potato plant itself grows best at a relatively low temperature (about 18° C.). The latter applies particularly to tuber development. Richards (21) states that the potato *Rhizoctonia* organism, *Corticium vagum*, does its greatest damage at a soil temperature between 15° and 21° C. with an optimum of about 18° C. The severity of the attack decreases rapidly above 21° until at 24° the fungus proves to be of minor parasitic importance and at 27° or above few typical lesions occur.



He found that 18° C. was also the optimum for host development. The same author (22) describes, in a later article, another piece of work with the same organism, but on other hosts, the pea and bean. He found that the organism does the greatest damage to both these hosts at an optimum soil temperature of about 18° while the pea plant itself does best at a temperature of 18° to 21° and the bean plant at 24° to 30°. Thus it will be noticed that in these experiments the fungus, *C. vagum*, has a constant optimum of 18° C. regardless of whether or not this temperature is optimum for the host plant. Richards' work would seem to indicate that the potato and pea are most susceptible to attack by *Corticium vagum* when growing under optimum soil temperature conditions for their own development, whereas the bean is most susceptible at temperatures below its optimum. In a study of the influence of soil temperature on the development of the seedling-blight of wheat and corn caused by *Gibberella saubinetii*, Dickson (4) found that the wheat plant develops best at a soil temperature of 16° to 20° C. for spring wheat and 12° to 16° for winter wheat. The maize plant is favored by a temperature of 24° to 28° C. The seedling-blight disease develops best on wheat at a soil temperature of 12° to 28°, and on corn at 8° to 20°. It will be noticed that the range for the development of the disease on wheat is higher than the range for the host, while on corn the disease is favored by a temperature lower than the optimum for the host plant. He checked his greenhouse work against field observations and found that sowing spring wheat early and winter wheat late checked the blight while planting corn late was also effective in lessening the injury from this disease. Hungerford (10) states that he secured the highest percentage of bunt infection in wheat at a soil temperature of 9° to 12° C. Clayton (2), working on the Fusarium wilt of tomato under greenhouse conditions, found the optimum soil temperature for the development of the disease to be 27° C. while at 17° or 35° the disease made little progress. Walker and Wellman (28) found that germination of the spores of the onion smut fungus, *Urocystis cepulae*, is inhibited at a temperature of about 28° or 29° C. Montieth (18, 19), working with the club-root of crucifers, showed that the severity of clubbing on the roots is directly correlated with the vigorous growth of the host, and that a soil temperature of 20° C. seems to be the optimum for host development as well as for the most severe manifestations of the disease. Tisdale (26) showed that the flax-wilt fungus, *Fusarium lini*, grows best in culture at a temperature of 26° to 28° C. The minimum temperature for growth is 10° and the maximum 37°. He found that the minimum temperature at which infection of flax plants will occur is about 15° and that the temperature at which the disease

is most destructive to flax plants corresponds closely to the optimum temperature for the growth of the fungus in culture. In experimental work on the relation of temperature to infection of cabbage by *Fusarium conglutinans*, Gilman (8, 9) found that a soil temperature of 17° to 22° C. or above induced the disease while no disease occurred in plants grown in soil held at temperatures below 17°. Johnson (11) states that the most important factor influencing the development of the tobacco root-rot caused by *Thielavia basicola* is soil temperature. This disease is of little importance at soil temperatures below 15° or above 30° C. and the optimum temperature for its development ranges from 17° to 23° C.

*Effect of soil moisture.* — The pathogenicity of many parasites is affected by the soil moisture almost if not quite as much as by the temperature of the soil. Clayton (3) found that tomato plants were most susceptible to attack by the *Fusarium*-wilt fungus when the moisture content of the soil was optimum for a vigorous and succulent growth of the host and that plants grown in a soil with either a very low or a very high moisture content were resistant or immune to the disease. Dickson (4) states that low soil moisture favors infection of wheat and corn seedlings by the seedling-blight fungus, *Gibberella saubinetii*. Hurgerford (10) secured the highest percentage of bunt infection in wheat sown in the soil with a moisture content of 22 per cent. The club-root disease of crucifers is favored by a soil moisture of 60 per cent or above, while a moisture content of 45 per cent or less inhibits the development of the disease (19). Sanford (23) found that the soil moisture apparently exerts as much influence as, if not more than, the soil reaction, upon the development of the common scab of potato. His experimental results show that potatoes grown in dry soil are more scabby than those grown in moist soil. For example, at a soil-moisture percentage of 14 per cent he secured a very scabby crop while at 34 per cent moisture content scarcely any scab developed. He checked up on the soil acidity and found that his dry-soil plots showed a slightly greater hydrogen-ion concentration than the moist-soil plots.

*Temperature and moisture of the air.* — As would naturally be expected, most of the work on the influence of air temperature and humidity on plant diseases has been done in connection with those parasitic diseases which are air-disseminated and attack the aerial parts of plants. However, there is evidence that even some of the soil-borne diseases may respond in some degree to the influence of air temperature. During recent years abundant evidence of the effect of air temperature on the visual manifestation of virus-disease symptoms has been accumulated. In addition to the direct effect of air temperature and humidity

on plant diseases attention has recently been called (24) to the apparent effect of the rate of transpiration, which of course depends in turn upon air temperature and humidity, and the velocity of wind movements.

Among the parasitic diseases there are numerous examples of the effect of air temperature and humidity. Many observations over a long period of years indicate that outbreaks of the late-blight of potatoes occur during seasons of excessive precipitation and sub-normal temperatures. (See p. 255 and references 7, 10, 12 under late-blight of potatoes.) As has been already noted, peach leaf-curl is more prevalent when the early spring weather is wet and cold. Apple-scab is a disease which develops well in cool, moist weather; consequently a rainy period just as the buds are opening favors an outbreak of this disease. Keitt and Jones (16) made a comparative study of apple-scab and cherry leaf-spot with reference to their environmental relations. They found a striking contrast in that even though the spores of the cherry leaf-spot fungus were present as early as those of the apple-scab fungus, there is no early development of the cherry disease because the causal fungus is a high-temperature organism and does not become active until warmer temperatures prevail. Thus the most serious apple-scab infections may occur before blossoming whereas no cherry leaf-spot infections of importance take place until after blossoming time. Giddings (6, 7) found a very close relation existing between weather conditions and the production and dissemination of the sporidia of the apple-rust fungus, *Gymnosporangium juniperi-virginianae*. The optimum temperature for infection is in the neighborhood of 65° F. Considerable precipitation is necessary to germinate the teliospores but apparently the sporidia are not dispersed until there is a decrease in humidity and some evaporation occurs following the rain. The apple-tree anthracnose of the Pacific Northwest is a good example of a fungous disease requiring considerable moisture. Few infections, if any, occur until after the autumn rains set in when the bark of the trees is kept continuously wet during long periods and the atmosphere is practically saturated with moisture. Lauritzen and Harter (17) found that the percentage of infection of sweet potatoes by *Rhizopus* varies greatly with the relative humidity. Only a very small percentage became infected at relative humidities of 93 to 99 per cent, while at a humidity of 75 to 84 per cent almost 100 per cent of infection occurred and at 51 to 52 per cent humidity very little infection occurred. Clayton (2) found a very interesting situation in his investigations on *Fusarium* wilt of tomato. As mentioned above, he discovered that a soil temperature of 27° C. favored the development of the wilt but only when the air temperature was also favorable (27°-33° C.). When the air was too cool, 17°, and the soil

optimum, heavy infection occurred in the root and basal part of the stem but the plants grew thriftily and showed no signs of the disease above ground. Among the virus diseases it has been found that the symptoms of many of them are masked by high temperatures while in one case at least the symptoms are accentuated by high temperatures. (See under virus diseases, Chapter XXIV.)

**Effect of environment on host versus effect on parasite.** — The question naturally arises as to whether these various factors of the environment exert a greater influence upon the host than upon the parasite or *vice versa*. This is a difficult question to answer but there seems to be evidence that in some cases the influence is exerted chiefly upon the host while in other cases it is chiefly the parasite which is affected. In many cases there is probably a mutual influence exerted upon both host and parasite. In case of the onion-smut caused by *Urocystis cepulae*, experimental work (27, 28) has demonstrated that the disease decreases with rising temperature and is meager at 27° C. This effect has been shown to be due to the influence of soil temperature upon spore germination which is inhibited at about 28° C. and above. Montieth (19) believes that the influence of high soil-moisture content upon cabbage club-root is due to the necessity of water for spore germination as well as for migration and infection. He found that a lower moisture content in the soil was needed for cabbage development than was required for the occurrence of the disease. On the other hand he found that the influence of temperature was exerted on the host rather than the parasite as shown by the larger clubbing of the roots in direct proportion to the amount of root growth. Dickson (4) judged that in case of the seedling-blight of wheat and corn, referred to above, the influence of temperature on the disease was exerted upon the host rather than upon the parasite. This is explained (14) as a case wherein the hosts are rendered susceptible by the adverse temperature for host development, and likewise resistant by temperatures favorable for the most rapid development of the host. In other words, the young embryonic tissues of the seedling are easily penetrable by the fungus, but when conditions are favorable for the development of the host seedling it rapidly passes from this susceptible condition to one of greater resistance.

Many other examples might be cited, but the foregoing are sufficient to emphasize the part played by different factors of the environment, both singly and in combination. It will thus be seen that these factors may easily determine either the regional distribution or the seasonal occurrence of a particular disease. For example, the bean anthracnose (*Colletotrichum lindemuthianum*) is of no consequence in Louisiana during the hot summer months although it is quite prevalent during the

cooler season. In this case the limiting factor is the temperature at which the growth of the fungus is inhibited (about 34° C.). In Oregon the powdery dry-rot of potato caused by *Fusarium trichothecioides* is prevalent in the arid section east of the Cascade mountains, while the dry-rot caused by *Fusarium coeruleum* is more common west of the Cascades. Apparently the moisture factor is the determining element in this case. Almost every year examples of the seasonal influence of weather on the distribution of plant diseases occur. This is evidenced by the fact that such diseases as wheat stem rust, late-blight of potato and apple-scab, occur in epidemic form during some seasons while they are practically absent in the same localities during other seasons.

**Effect of environment on non-parasitic diseases.** — It is the general opinion that most, if not all, of the well-known non-parasitic diseases are due largely, if not entirely, to the environment. While there may not be general agreement as to the exact manner in which such diseases are produced, it is conceded that moisture, temperature, and soil condition, singly or in combination, are responsible for these troubles. For example, many specific theories have been advanced to account for bitter-pit in apples. The details of these theories differ but all of them agree on one point, namely, that some disturbance of the water balance in the plant causes the trouble. Cork and drouth-spot are also attributed to a disturbed water balance. Point-rot of tomato is another disease supposed to be due to the same general cause. It can readily be seen that weather conditions play an important part in diseases of this kind, but it is not difficult to conceive that the physical condition of the soil might also be a factor in some cases mentioned above. In cases such as apple-scald, ventilation plays an important part. Improper temperatures are largely responsible for black-heart of potatoes, though good ventilation is also desirable in avoiding this trouble. A more complete discussion of some of the non-parasitic diseases will be found in Chapter XXV.

#### REVIEW QUESTIONS

1. Define "ecology."
2. Under what two chief heads may the factors of the environment of plants be listed?
3. Name three factors of the soil environment.
4. What are the main elements of the meteorological factor?
5. What is meant by the "biological condition" of the soil?
6. How is the biological condition of the soil related to plant disease?
7. Cite examples of the influence of the chemical composition of the soil upon plant diseases.
8. Cite examples of the influence of soil temperature in limiting the geographic distribution of plant diseases.

9. Is soil reaction the only environmental factor influencing the development of common scab of potato? (See reference 23.)
10. What other factors besides soil reaction affect the occurrence of cabbage club-root? (See reference 19.)
11. Cite examples in which the influence of environmental factors is exerted more upon the parasite than upon the host, and *vice versa*.
12. In general, what factors are concerned in influencing soil-borne diseases as compared with those diseases which are disseminated through the air and attack the aerial parts of plants? Cite examples of each.

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## CHAPTER VI

### PLANT DISEASE CONTROL: GENERAL STATEMENT

Historically man has always manifested an interest in the control of plant diseases as well as in their cause. The practical agriculturist, of course, is primarily interested in protecting his crops from diseases and usually cares little about the cause of the trouble provided he can find an efficacious remedy. The latter statement is true until he is made to realize that the most efficient control measures usually are based on accurate knowledge of the nature of the trouble. In early times the remedies recommended were necessarily empirical since nothing was known of the cause of disease in most cases. Even today we are using many control measures that originated empirically, however much we may have modified and adapted them in recent years in accordance with our increased knowledge of the cause of the disease. With the gradual acquisition of more accurate information on the nature and cause of diseases we have worked out methods and devices to meet the special need in combating any particular disease.

Before entering upon a discussion of the various methods used in controlling different types of plant diseases attention should be called to factors which may make it necessary to modify the procedure followed in combating a particular disease, depending upon the conditions under which one is working. Perhaps the most important factor here concerned is that of environment. After reading Chapter VI on the relation of environment to plant disease the reader will have a basis for readily understanding why it is not necessary or desirable to apply exactly the same control program to a particular disease under any and all conditions. For example, in applying a spray program for the control of apple-scab the number of applications of spray and the time at which each application is made will depend upon the weather and the stage of development of the new growth on the apple tree. In localities or in seasons where the spring and early summer weather is rainy a larger number of applications will be necessary to control the disease than in a locality or season where the weather at the time the buds open and continuing through the spring and summer is exceedingly dry. In the former case perhaps five, six or more applications of spray may be necessary to control the disease, whereas in the latter case possibly only one, two or three applications may be sufficient, and in



some cases perhaps no spraying at all will be necessary. Another illustration of this principle is found in the club-root of cabbage. A frequently recommended control measure for this disease is the application of lime to the soil. But is the grower to apply lime blindly to all land upon which he expects to grow cabbage? Not at all. The principle upon which the above recommendation is made is the apparent fact that an acid soil favors the development of the club-root disease and that lime added to the soil will correct the acidity and thus render such soil less favorable for the growth of the club-root organism. If the soil is not acid there would be nothing gained by adding lime so far as the control of cabbage club-root is concerned.

Any number of examples could be cited illustrating the above principle but there is neither space nor necessity for doing so. In reading the remainder of this chapter and the following chapters on various phases of plant-disease control as well as the control measures recommended under each disease discussed in Part II, the reader is urged to keep this principle constantly in mind. Very few recommendations for plant-disease control, if any, should be followed blindly and literally in all cases and under all circumstances. The grower must study local conditions with regard to soil, weather, climate and the occurrence of diseases in his locality and adapt control measures to meet the circumstances. The student should study every disease taken up in this course from this point of view before making definite recommendations for controlling it.

It would be difficult to compile a complete list including all the different methods with their various modifications and adaptations which have been devised to control all the multitude of plant diseases with which the agriculturist has to contend. However, an attempt has been made to make as complete and as specific a list as possible. The control methods outlined below are grouped, for the sake of convenience, according to the type of disease (that is, parasitic, virus, non-parasitic) for which the measures are recommended. Examples are given after each method illustrating the types of diseases to which the different control measures are applicable.

#### I. Control of parasitic diseases.

##### 1. Use of fungicides.

###### a. Applied to the host.

(1) *Spraying*. — Apple-scab, peach leaf-curl, and late-blight of potatoes are examples of diseases which are combated chiefly by spraying with a fungicide to protect susceptible parts from infection.

(2) *Dusting.* — Dusts are applied for the same purpose as sprays in cases where a fungicide in dust form will protect as effectively as in liquid form or where for some reason a liquid spray is not feasible. Dusts are effective against grape powdery mildew and in some cases give more or less satisfactory control of various other diseases.

(3) *Disinfection of seed or other propagating stock.* — Seed potatoes are disinfected with various fungicides, as formalin or corrosive sublimate, for such diseases as scab and Rhizoctonia. Seed grain is disinfected for smut with either liquid or dust fungicides.

(4) *Disinfection of wounds.* — In cutting out fire-blight a disinfectant is used to prevent spreading the bacteria to the new wounds where re-infection would occur. Large wounds of any sort on a tree should be covered with a fungicide to prevent entrance of wood-rotting fungi.

b. Applied to the environment.

(1) *Soil sterilization.* — Soil may be sterilized in various ways, as by the application of steam, hot water, formaldehyde or sulfuric acid, to kill damping-off fungi, eelworms or other pathogenic organisms living in the soil.

(2) *Sterilization of tools.* — In cases where resort is had to cutting out diseased parts of plants, particularly with certain bacterial diseases like fire-blight, it is recommended that the tools be sterilized between cuts to prevent carrying the germs from diseased tissue and depositing them on the healthy tissue left exposed by the cut.

(3) *Disinfection of containers, etc.* — In cases where the pathogene in the form of spores or bacteria separates from the host and adheres to containers, carriers or storage quarters there is danger of contaminating the next batch of products placed in such containers; therefore the containers or carriers should be disinfected before filling again. This will apply to grain sacks which have held smutty grain, boxes or crates in which produce has molded or decayed, potato storage quarters and railway cars.

2. Use of disease-free seed or other propagating stock.

a. *Selecting clean seed from a mixed lot.* — Where disease on seeds can be detected readily enough it is sometimes possible to sort out the diseased seeds and separate them from the healthy ones. Bean seeds affected with anthracnose or potato seed tubers affected with scab may be sorted out in this way. However, as a rule such sorting is not satisfactory because of the impossibility of detecting and eliminating all diseased seed. Some slightly affected ones will almost certainly escape notice so that this method is usually not satisfactory.

*b. Securing seed from a field or locality where no disease exists.* — It is much safer to secure bean seed from a field or locality where no disease occurs than to try to sort a diseased lot. The same will hold in general for seed potatoes.

*c. Use of a special seed plot.* — It is a good plan to maintain a special plot for growing sufficient seed of beans, potatoes or any desired crop to plant the commercial plantings from year to year. This plot should be well isolated from other fields and a special effort should be made to keep it clean.

3. *Crop rotation.* — Many pathogenic organisms harbored in the soil are partial to certain species of crop plants. If these certain crops are not grown in a field for a few years the special parasites of the crop will die out in that field. It will then be safe to grow the susceptible crop again on this soil. The length of time an organism will survive in the soil in the absence of its host plant determines the length of the rotation cycle.

4. *Surgery (cutting out diseased parts).* — Many of the canker diseases such as fire-blight, European canker and apple-tree anthracnose can be fought more or less successfully by cutting out the diseased areas.

5. *Roguing (uprooting and removal of diseased plants).* — This method is practiced with truck and vegetable crops such as potatoes. It can be successfully used against those diseases which can be recognized in the field during the growing season, as, for example, the wilt diseases of potatoes.

6. *Removal of alternate or complementary host.* — Many of the rust fungi are heteroecious, that is, they complete their life cycle on two types of hosts, as, for example, the stem rust of wheat and the white-pine blister-rust. One means of combating the wheat rust is to eradicate its alternate host, the barberry. Likewise the white-pine blister-rust can be fought by destroying the complementary hosts, currants and gooseberries.

7. *Removal of weed hosts.* — Many parasites causing diseases of economic plants also attack other related plants which are weeds or of no economic importance. Examples of this are the fire-blight organism which attacks the hawthorn, service-berry and wild crab in addition to the cultivated pomaceous fruits, and the *Rhizoctonia* fungus which attacks a great variety of plants, including some common weeds. The destruction of these weeds or non-economic hosts is to be recommended as one means of combating these diseases.

8. *Sanitation (destruction of diseased plants or parts of plants).* — A great many plant-disease organisms winter over, or are perpetuated for a short time at least, on various forms of plant debris. The brown-

rot and bitter-rot fungi hibernate on rotted and mummified fruits. The late-blight of celery overwinters on the trimmings from diseased plants which are left in the field. The fire-blight organism survives for a time in prunings which are left on the ground. All such diseased débris should be burned or destroyed in some way.

9. *Changing the soil reaction.* — The acidity or alkalinity of the soil sometimes exerts a strong influence upon plant pathogenes which live in the soil or attack the under-ground parts of plants. If a certain organism thrives best in an acid soil it may be combated by changing the reaction of the soil by the addition of lime, for example. If it is an organism which is favored by an alkaline soil the control procedure may be reversed and the soil treated to render it less alkaline. (See Chapter V.)

10. *Resistance and immunity.* — Some varieties of plants are less susceptible to a specific disease than other varieties, as, for example, some varieties of wheat are more resistant to rust or to smut than other varieties. Other things being equal, the more resistant variety should be grown. (See Chapter XI.)

11. *Insecticides and insect repellents.* — Some diseases are disseminated by insects. Fire-blight is a good example of this. Bacterial wilt of cucurbits is another. If the insects which carry these disease germs can be killed or repelled, the spread of the disease in this manner may be prevented. (See Chapter XII.)

12. *Quarantine and inspection.* — This method of control is used to prevent the introduction of a disease from a country or section of a country where it is prevalent into a region which is free from the disease. Some diseases against which quarantines have been established at various times and places are the white-pine blister-rust, potato wart and the citrus canker.

## II. Control of virus diseases.

1. *Use of disease-free seed or other propagating stock.* — Bean mosaic is said to be carried in the seed, but most of the mosaic diseases so far as known at present are perpetuated in vegetative parts of plants which are used for propagating purposes, such as tubers, bulbs, cuttings and sprouts. All of this propagating stock of whatever nature should be selected from disease-free plantings. (See Chapter VIII.)

2. *Insect control.* — Many of the virus diseases are known to be transmitted by insects, principally aphids and leaf-hoppers. In so far as practical or feasible the control of these insects will lessen the spread of the virus diseases which they transmit.

3. *Destruction of weed hosts.* — Many of the virus diseases are now known to be perpetuated in weed hosts, as, for example, tomato

mosaic in horse nettles and cucumber mosaic in milk weeds. They are then carried from these weeds to the cultivated crops by insects.

4. *Tuber and eye index.* — The growing of one tuber from a hill or one eye from a tuber gives promise of being a practical method of determining whether the remainder of the tubers in the hill or the remainder of the eyes in a tuber will produce virus-free progeny.

### III. Control of non-parasitic diseases.

#### 1. Modifying the environmental factors.

a. *The physical condition of the soil.* — In so far as the physical condition of the soil has to do with its water-holding capacity it is a factor in the cause of those non-parasitic diseases which are due to lack of the proper water balance. Changing this condition of the soil by proper cultural methods may aid in preventing certain non-parasitic diseases. (See Chapter V.)

b. *Moisture supply.* — Wherever this factor can be controlled, as on irrigation projects or in the greenhouse, it can sometimes be manipulated so as to prevent certain non-parasitic diseases, as for example, bitter-pit of apple or blossom-end-rot of tomatoes.

c. *Temperature.* — Black heart of potatoes is due in part at least to unfavorable temperatures in storage. In so far as this is true the correction of this unfavorable temperature will help to prevent this disease.

d. *Ventilation.* — Apple-scald is a non-parasitic disease which may be satisfactorily controlled by proper ventilation.

The above is not a hard and fast classification of control measures nor is it necessarily a complete list. It will be easy to find some overlapping in the various groups. For example, "modifying the environmental factors" might be listed under the control of parasitic diseases as well as of non-parasitic, since unquestionably the environment is related to parasitic diseases also. The use of resistant or immune varieties is a means of control that can be applied, to a certain extent, to all three types of diseases mentioned in this outline. Some writers (2) generalize and list all control methods under four general headings, namely (a) exclusion, (b) eradication, (c) protection and (d) immunization. It would make an instructive exercise for the student to rearrange all the control methods given in the detailed outline above under these four headings.

There is no intention of discussing at length, at this point, every one of the different items in the detailed outline above. However, in the succeeding chapters of Part I several different phases of the control problem, or topics related to control, will be discussed in some detail. Also, as previously suggested, applications of various specific methods

of control are made under each disease listed for discussion in Part II. In making a detailed study of any disease it is suggested that the student take note as to which of the methods mentioned in this chapter can be applied to the control of the disease in question and why this is so.

### REVIEW QUESTIONS

1. Name twelve different control measures which may be used against various parasitic diseases.
2. Mention the different uses of fungicides in controlling plant diseases.
3. What are the various means of securing disease-free seed?
4. Under what circumstances will crop rotation control a disease?
5. What is meant by "roguing"? When may this method be used effectively?
6. With what type of disease may eradication of an alternate host be effective?
7. What is the difference between a weed host and an alternate host?
8. What is meant by sanitation with reference to plant-disease control?
9. Under what conditions may correcting the acidity or alkalinity of the soil aid in controlling a disease?
10. What relation does the use of insecticides and insect repellents bear to plant-disease control?
11. Under what circumstances is a quarantine advisable as a control measure?
12. What methods lend themselves to the combating of virus diseases? Why?
13. Under what one head may all of the measures which may be used in combating the non-parasitic diseases be listed?
14. List all of the control measures discussed in this chapter under four general heads.

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## CHAPTER VI

### FUNGICIDES

A fungicide is any agent of whatever nature that is applied either to the plant or plant parts, or to the environment of the plant for the purpose of disinfecting the plant or plant parts, or the environment, or for the purpose of protecting the plant against infection by parasitic fungi or other organisms. Fungicides may be classified on two bases, namely, (a) nature of the fungicide, and (b) purpose for which used. The following outline indicates the classification of some of the principal fungicides on these two bases.

#### I. On basis of nature.

##### A. Chemicals.

##### 1. Copper-containing fungicides.

###### a. Copper sulfate.

(1) In dust form.

(2) In solution.

###### b. Copper sulfate plus lime.

(1) Bordeaux mixture.

(2) Bordeaux paste.

(3) Copper sulfate dip plus lime-water bath.

(4) Copper-lime dust.

###### c. Copper sulfate plus sodium carbonate.

(1) Burgundy mixture.

###### d. Copper carbonate.

(1) Dust.

(2) Ammoniacal copper carbonate.

###### e. Copper acetate.

##### 2. Sulfur fungicides.

###### a. Lime-sulfur.

###### b. Commercial dry lime-sulfur.

###### c. Self-boiled lime-sulfur.

###### d. Wettable sulfur (sulfur paste).

###### e. Cold-mix lime and sulfur.

###### f. Dry-mix sulfur-lime.

###### g. Sulfur dust with or without insecticide or spreader.

3. Iron sulfate.
  4. Potassium sulfide.
  5. Formaldehyde.
  6. Bichloride of mercury.
  7. Cyanide of mercury.
  8. Organic mercury compounds.
  9. Creosote.
  10. Zinc chloride.
  11. Sulfuric acid.
  12. Calcium hypochlorite.
- B. Heat.
1. Hot water.
  2. Steam.
  3. Dry heat.
- II. On basis of purpose for which used.
- A. For protection.
1. Applied to the growing or dormant plant.
  2. Applied to wounds.
  3. Wood preservatives.
- B. For disinfection.
1. Applied to seed or propagating part.
  2. Applied to the environment.

Many different chemicals have been tried out as fungicides but relatively few of them have proved effective enough to warrant general adoption. Some of the more effective ones are discussed briefly in the succeeding pages.

1. **Copper-containing fungicides.** — As noted in Chapter I the use of copper in the form of copper sulfate (bluestone) was practiced at an early date. Prevost used it to disinfect seeds in 1807. The persistent and continued use of copper fungicides, however, dates from the discovery of bordeaux mixture by Millardet about 1883. Copper sulfate, as an ingredient of bordeaux or used alone, still remains the most important and most extensively employed compound of copper in the treatment of fungous diseases of plants, but it is not the only copper fungicide. The use of copper carbonate is increasing in recent years, particularly in the dry treatment of seed wheat for stinking smut. Also, it is used sometimes in making a substitute for bordeaux mixture.

**Copper sulfate.** — Copper sulfate alone is quite commonly used as a disinfectant for seed or for containers, storage quarters, etc. It is customary to use it in solution for these purposes although in some cases attempts have been made to use it in dry or powdered form. For the



treatment of seed wheat for smut the following formula is commonly employed:

Copper sulfate (bluestone).....	1 lb.
Common salt.....	1 lb.
Water.....	5 gals.

A lime bath is usually given the seed after this treatment. A dust mixture of equal parts of copper sulfate and air-slaked lime has been used as a seed disinfectant for bunt of wheat with poor success. For disinfecting potato bins for rots the following formula is recommended:

Copper sulfate.....	1 lb.
Water.....	10 gals.

A solution of copper sulfate in water at the rate of 2 to 3 lbs. per 50 gals. of water is sometimes used as a spray. It is safe only on dormant trees. It has been used with some success for peach leaf-curl but is not as permanent as bordeaux since it readily dissolves away during rainy weather. It may be said that copper sulfate has not given perfect satisfaction when used for the above purposes, especially in treating seed wheat, and it seems probable that it will ultimately be replaced by more satisfactory fungicides, some of which will be discussed later.

**Bordeaux mixture.** — Bordeaux mixture is one of the two most important sprays in use at the present time. For certain diseases it is much more effective than lime-sulfur, its rival. Where a spray is needed which has the sticking and lasting qualities required to give protection over a long period of time, as is the case with such diseases as Northwestern anthracnose of apple and *Coryneum* peach blight, nothing has yet been found to equal bordeaux mixture. Also in certain diseases such as late blight of potato, bordeaux gives much better results than lime-sulfur and does not cause injury to the host. The ingredients of bordeaux mixture are copper sulfate, lime and water. The varying proportions of these components give rise to the different formulae in use. The 4-4-50 formula follows:

Copper sulfate.....	4 lbs.
Stone lime.....	4 lbs.
Water.....	50 gals.

For spraying potatoes the strength is usually increased to 5-5-50 or 6-6-50. For use on more delicate plants it is necessary to make a weaker spray, for example, 2-4-50 or 3-6-50. Other formulae may be used if advisable.

*Directions for making bordeaux.* — In making bordeaux mixture solutions of the bluestone and of the lime should be made up separately. For example, if the 4-4-50 formula is to be used, dissolve 4 lbs. of copper sulfate in 25 gals. of water. Bluestone dissolves very slowly in cold water; use hot water, or suspend the bluestone in a gunny sack from a stick placed across the top of an open wooden barrel so that the material is barely below the surface of the water. Do not dissolve the copper sulfate in a metal vessel. Slake 4 lbs. of lime in another vessel, add the water slowly and when the lime is completely slaked add enough water to bring it up to 25 gals. When ready to use, pour these two solutions together while stirring.

When large quantities of spray are needed it is better to make up large quantities of more concentrated solutions of copper and lime. This may be done by dissolving 50 lbs. of copper sulfate in 50 gals. of water and slaking 50 lbs. of quick lime and making up to 50 gals. with water. This gives a stock solution of each containing 1 lb. of bluestone or lime to the gallon. Then if, for example, 200 gals. of 4-4-50 spray are needed, strain 16 gals. of the lime solution into the tank after having filled the tank two-thirds full of water. Stir thoroughly, adding 16 gals. of the bluestone solution which has been somewhat diluted. Finally fill the tank up to 200 gals. This gives a 200-gallon tank full of 4-4-50 bordeaux. The concentrated solutions of copper sulfate and lime should never be mixed before diluting as this gives a very inferior quality of spray material.

*Sugar as a preservative.* — Bordeaux mixture deteriorates very rapidly upon standing. If for any reason a quantity of spray is made up and cannot be used within a few hours, it may be preserved in good condition for an indefinite time by adding a small amount of common sugar, 1 oz. for every 8 lbs. of stone lime or 10 lbs. of hydrated lime used in the spray.

*Hydrated lime a substitute for stone lime.* — Under some conditions it may be desirable to use hydrated lime instead of stone lime in making up bordeaux. In such cases it is necessary to use one-third to one-half more of the former than would be used of the latter. For example, to make 4-4-50 bordeaux one must use  $5\frac{1}{2}$  to 6 lbs. of hydrated lime instead of the 4 lbs. of stone lime for each 50 gals. of spray. The quality of hydrated lime bordeaux is never quite equal to that made from stone lime.

**Bordeaux paste.** — One of the best dressings that has been devised to protect tree wounds against infection by wood-rotting fungi is bordeaux paste (16). This contains the same ingredients as bordeaux mixture but with much less water in proportion to copper and lime. The usual formula follows:

Copper sulfate,  $1\frac{1}{2}$  lbs. in 1 gal. of water.

Quick lime, 3 lbs. slaked in 1 gal. of water.

Mix in equal parts.

This makes a paste which is applied to the wound in the same manner as white-wash. For dissolving the copper sulfate see directions under bordeaux mixture. A satisfactory bordeaux paste can be made from some of the dry bordeaux powders on the market, by bringing to the consistency of white-wash by the addition of water and applying as above. A more permanent bordeaux paint can be made by using raw linseed oil (18) instead of water with the dry bordeaux. Add the oil slowly while constantly stirring until a paste is secured of the right consistency to apply with a paint brush. One application of this preparation will remain effective for several seasons.

**Copper-lime dust.** — In 1918, Sanders and Kelsall (1, p. 286) began some experimental work in Nova Scotia on a type of copper dust for which they later claimed marked success. Their formula follows:

Dehydrated copper sulfate . . . . .	12 lbs.
Arsenate of lime . . . . .	10 lbs.
Hydrated lime . . . . .	78 lbs.

In a two-year comparative test it was claimed that the copper-lime dust was equally as effective as bordeaux for the control of late blight of potato.

**Burgundy mixture.** — This is another substitute for bordeaux and may be used to avoid staining or undesirable deposits on fruit and foliage. The formula follows:

Copper sulfate . . . . .	1 lb.
Sodium carbonate . . . . .	$1\frac{1}{2}$ lbs.
Water . . . . .	50 gals.

Dissolve each chemical separately as in making bordeaux mixture. Make up the stock solutions at the rate of 1 lb. to 1 gal. Before mixing the two dilute the copper sulfate with the requisite amount of water, then add the sal soda solution.

**Copper carbonate.** — Powdered copper carbonate is rapidly coming into prominence as a disinfectant for bunt of wheat. It is used at the rate of 2 ozs. of the dust per bushel of wheat. One great advantage of this dust is that it does not reduce the stand as is so often the case with the formaldehyde and bluestone treatments. Its effectiveness, however, depends on the fineness of the powder, the amount of copper present, and the thoroughness with which each grain is coated, a machine being necessary for effective dusting of seed wheat.

**Ammoniacal copper carbonate.** — In spraying fruits nearing maturity, with bordeaux mixture, an unsightly deposit is usually left on the fruit. In order to avoid this undesirable result this spray was devised. It is not usually as effective as bordeaux but in some cases it may be desirable to use it in order to escape the bad effects of bordeaux. The usual formula follows:

Copper carbonate.....	5 or 6 oz.
Ammonia.....	3 pts.
Water.....	50 gals.

To prepare this spray first dissolve the copper carbonate in the ammonia and then add the water.

**Copper acetate.** — While this form of copper has never come into prominence as a fungicide, Butler (7) claims that it has great possibilities. In this connection it should be said that there is a great field open for investigations looking toward the finding of better fungicides than the few now in common use, as well as the accumulation of more complete information concerning the standard materials now relied upon.

**2. Sulfur-containing fungicides.** — Sulfur as a remedy for certain diseases, especially powdery mildews, has been known for over a century (14).

**Lime-sulfur.** — In addition to its use in the dust form, sulfur is combined with lime to make the well-known lime-sulfur sprays of which there are several variations. Following are formulae for making concentrated lime-sulfur.

Sulfur.....	2 lbs.
Quicklime (stone lime).....	1 lb.
Water.....	1 gal.

Lime-sulfur can be made up in any quantity according to need and facilities for making, the ingredients being used in approximately the proportions of the above formula. To make 50 gals. of concentrate use:

Sulfur.....	100 lbs.
Quicklime.....	50 lbs.
Water.....	50 gals.

Small quantities may be made in a kettle over an open fire. In this case one has to use a slight excess of water to make up for loss by evaporation. If facilities are available large quantities can best be made by steam cooking. A pipe should be run from a steam boiler directly into the cooking tank and steam turned into the mixture. The cooking should continue for about 45 minutes over the fire or about 30 minutes

if steam is used. Settle and draw off the clear liquid, discarding the sludge. It is usually possible to purchase the concentrated solution ready for use. Where this can be done it is for the grower to decide whether it is more convenient and economical to purchase the commercial solution or to prepare it at home. In any case before the spray is used it is necessary to determine the strength of the stock solution so as to know how much to dilute it for the various applications. A Baumé hydrometer should be purchased from the druggist and used for this testing.

The following table quoted from Oregon Exp. Sta. Bul. 201 (13) explains how to prepare the different strengths of spray depending upon the hydrometer or specific-gravity test of the concentrated stock solution.

DILUTION TABLE FOR LIQUID LIME-SULFUR

Strength of stock solution		To make 100 gals. of dilute spray use the number of gallons of concentrated stock lime-sulfur indicated in the columns below and add water to make 100 gals.				
Degrees Baumé	Specific gravity	1 Dormant strength for scale clean-up (12-100)	2 Dormant strength for blister-mite and twig-miner (8-100)	3 Early spring spray (3½-100)	4 Mid-spring spray (2½-100)	5 Late spring spray (2-100)
°		gal.	gal.	gal.	gal.	gal.
34	1.304	11+	7½	3+	2½+	1¾+
32	1.282	12	8	3½	2½	2
30	1.260	12¾+	8½	3½	2½+	2+
28	1.239	14-	9½	3¾	2¾+	2¼+
26	1.218	15	10	4	3	2½
24	1.198	16½-	11	4½-	3½+	2¾
22	1.179	18¼+	12¼	4¾+	3¾-	3+
20	1.160	20¾+	13¾	5½-	4½-	3½

Note: Where the + sign is used, employ a little over the number of gals. indicated. Where the - sign appears use scant measure.

**Commercial dry lime-sulfur.** — For several years it has been possible to buy on the market a preparation known as "dry lime-sulfur." This product is made by the dehydration of concentrated lime-sulfur solution. It comes in the form of a powder which is readily dissolved in water. It has the advantage of being less weighty and less expensive to transport and is easily prepared by dissolving the required number of pounds in the requisite amount of water. Very good results may be obtained with dry lime-sulfur as a substitute for ordinary lime-sulfur.

Chemical analysis shows that about 4 lbs. of the dry lime-sulfur preparation must be used to give the same effective constituents as a gallon of liquid lime-sulfur of the standard 32° Baumé strength. For example, in making up 100 gals. of ordinary lime-sulfur spray of the 12 to 100 strength, 12 gals. of concentrated lime-sulfur are required for each 100 gals. of spray. If dry lime-sulfur should be substituted for the concentrated solution, 48 lbs. would be needed to make 100 gals. of spray. If weaker dilutions of dry or liquid lime-sulfur are used the grower may often obtain good control but the margin of safety is reduced.

**Substitutes for concentrated lime-sulfur.** — The regular concentrated lime-sulfur is known to cause injury of various kinds, especially in warm weather. Bordeaux mixture also causes injury under certain conditions. To obviate this injury or for other reasons, various sulfur substitutes are available. Among these substitutes for the standard lime-sulfur and bordeaux sprays are: self-boiled lime-sulfur, wettable sulfur, dry-mix sulfur lime and cold-mix lime and sulfur.

**Self-boiled lime-sulfur.** — For spraying peaches or other crops having sensitive foliage this specially prepared solution of lime-sulfur was devised some years ago by the U. S. Department of Agriculture. In making self-boiled lime-sulfur no heat is used except that generated by the slaking lime. This results in only slight chemical action between the sulfur and lime and makes a solution that is much safer to use on tender foliage. The usual formula follows:

Sulfur.....	8 lbs.
Quicklime.....	8 lbs.
Water.....	50 gals.

Start the lime slaking by adding a small quantity of the water, about 2 gals. As the lime slakes mix the sulfur with it. Add water as needed. As soon as the boiling ceases add enough water to cool the mixture, strain and dilute to 50 gals. This gives a spray that is largely a mechanical mixture of lime and sulfur rather than a chemical combination as is true with the regular lime-sulfur described above. It is disagreeable to make and is now largely replaced by the materials described in the paragraphs immediately following.

**Wettable sulfur (sulfur paste).** — Many substitutes for self-boiled lime-sulfur have been devised. They all consist of sulfur either alone or in mechanical mixture with lime or other substances. The active ingredient in all these substitutes is sulfur. Ordinary sulfur is not easily wetted and therefore is difficult to make up in the form of a spray. To overcome this difficulty several brands of so-called "wettable sulfur"

or "sulfur paste" have been manufactured and offered on the market under such names as "atomic sulfur" and "colloidal sulfur." These pastes are readily diluted in water to make a spray solution. Sulfur sprays of this sort have about the same fungicidal value as sulfur applied in the dust form. A grower can make a wettable sulfur for himself by using a finely divided grade of sulfur and gradually stirring into it a solution of calcium caseinate or sodium caseinate until a smooth paste is formed. This paste may then be diluted as desired for spraying.

**Dry-mix sulfur-lime.** — The New Jersey Experiment Station (8) has developed a substitute for self-boiled lime-sulfur which is claimed to eliminate some of the difficulties in the preparation of the latter. It consists of:

Sulfur.....	8 lbs.
Hydrated lime.....	4 lbs.
Calcium caseinate.....	8 ozs.

These ingredients are thoroughly mixed together while dry, then diluted with 50 gals. of water. Care must be taken to get the dry material wet thoroughly and evenly distributed throughout the diluted spray.

**Cold-mix lime and sulfur.** — Robinson (13) of the Oregon Experiment Station has recently developed still another substitute for the self-boiled lime-sulfur spray, which he calls "cold-mix lime and sulfur." To make 50 gals. of this spray use:

Sulfur (superfine flour).....	8 lbs.
Hydrated lime.....	4 lbs.
Skim milk.....	2 qts.

Mix the sulfur and hydrated lime together dry. Add 2 qts. of water to the 2 qts. of milk. Pour this into the sulfur and lime and stir to form a smooth paste. Add more water if necessary. Add several gallons of water and pour through a strainer into the spray tank. When made up to 50 gals. the spray is ready to use.

**Sulfur dust.** — Since about 1912 there has arisen great interest in the possibilities of sulfur as a fungicide when used as a dust. During the years from 1912 up to the present time a vast amount of experimentation has been done to determine the value of sulfur dust as compared with the liquid spray.

**Properties of good dusting material.** — The two prime qualifications of any fungicide which is to be applied as a dust are fineness and lightness. Formerly flowers of sulfur, a very fine sublimed sulfur, was the best available, but now it is possible to get finely ground sulfur which is much better than the flowers. In order to keep these fine forms from

"caking" or forming lumps, it is necessary to use some filler. It has been found that arsenate of lead dust forms an excellent filler, improving the dusting qualities of the sulfur, and at the same time makes a good combination insecticide. The proportions of sulfur and arsenate generally used in this combination dust are 90 parts of sulfur to 10 parts of lead arsenate, or 85 parts of sulfur and 15 parts of the arsenate. This combination dust is by far the most popular form of dusting sulfur, although it may be applied occasionally as pure sulfur without any filler of any kind. Hydrated lime is often used as a filler when an insecticide is not needed.

*Dusting versus spraying.* — During the last ten to fifteen years there has developed among plant pathologists, entomologists and horticulturists, a great interest in the feasibility of using dusts instead of liquid sprays. In 1914 Blodgett (4) said: "The use of finely ground sulfur as the fungicide in a dust mixture has given very encouraging results (on apples) and warrants further trials on a more extensive scale." In 1916 Stewart (15) in New York found that for leaf diseases in nursery stock "finely ground sulfur in the dust mixture proved as efficient a fungicide as lime-sulfur solution." In 1918 Giddings (10) stated that dusting did not prove satisfactory in West Virginia apple orchards, black rot, leaf spot and cedar rust not being appreciably controlled. In 1919 Fromme and Ralston (9) in Virginia found that sulfur-arsenate dust controlled peach scab but was unsatisfactory for brown rot. In Connecticut (1922) Britton (5) and others stated that they compared dusting with spraying for apple pests and diseases and in nearly all cases obtained the best apples from sprayed plots. On peaches, however, the dusted plots give slightly better fruit than the sprayed plots. The above are only a few examples of experiments on dusting for disease control. There is a large accumulation of literature on the subject and as shown above there is lack of accord as to the value of dusts as compared with sprays. The facts are that there are advantages and disadvantages in both and that probably neither can profitably be used to the exclusion of the other in all cases. The matter can probably best be summarized by making a list of the advantages as well as the disadvantages of the dust fungicides.

*Advantages.* — (a) Requires less time for application. Some claim it takes only one-fifth as much time as spraying. (b) Requires no water. This is quite an item when water is scarce or not near at hand. (c) Labor cost of application is less. (d) Enables the grower to get a fungicide on in short order during critical periods or uncertain weather conditions. (e) The initial cost of the dusting outfit is less than that of spray outfits of corresponding kinds.



**Disadvantages.** — (a) Material costs more per acre than sprays. (b) In many cases dusts are not as effective as sprays. (c) Dusts are more difficult to apply satisfactorily in windy weather.

**Stickers and spreaders.** — Various substances are added to sprays to make them adhere more satisfactorily to plant surfaces or to make them spread more evenly over the surface of fruit, foliage or bark. Some materials that have been used for this purpose are resin fish-oil soap, resin sticker, casein or calcium caseinate, oil emulsions and iron hydroxide (1, pages 16, 17, 64, 65).

3. **Iron sulfate.** — While this chemical is not a good fungicide in itself, it is sometimes used in connection with lime-sulfur for certain purposes. When added to lime-sulfur it forms a black precipitate which contains precipitated sulfur, iron sulfide and calcium sulfate. This preparation is said to have good fungicidal properties when used against apple powdery mildew where lime-sulfur cannot safely be used because of climatic factors as in the Pajaro Valley, California (2). The precipitated sulfur is the active fungicide in this mixture. Special care has to be observed in the preparation of this spray to avoid deleterious effects. For details see U. S. D. A. Bulletin 120, 1914. Another use that is sometimes made of iron sulfate, is as an indicator in the use of lime-sulfur. Some operators of spray outfits add a little iron sulfate to the lime-sulfur to give it a black color. This enables the man with the nozzle to see more readily how well he is covering the fruit and foliage. It is also claimed that it reduces the burning qualities of lime-sulfur and makes it more adhesive. Only a very little iron sulfate is needed when used as an indicator. One-half pound of iron sulfate for each gallon of concentrated lime-sulfur solution is sufficient. At this rate the following is a convenient formula:

Iron sulfate.....	2 lbs.
Dilute (1-50) lime-sulfur.....	200 gals.

Dissolve the iron sulfate in a little water or dilute spray and add to the tank in the above proportions.

4. **Potassium sulfide.** — This is an old-time remedy against certain fungus diseases such as powdery mildew of gooseberry. It is used as a spray made up after the following formula:

Potassium sulfide.....	3 oz.
Water.....	10 gals.

5. **Formaldehyde.** — Formaldehyde is used chiefly as a disinfectant for seed, soil, containers, bins, etc. It was formerly used quite exten-

sively for treating seed wheat for bunt. For this purpose the formula in common use is:

Formaldehyde (37 to 40 per cent strength) . . .	1 pt.
Water . . . . .	40 gals.

At present formaldehyde is not used as extensively for treating seed wheat as formerly. A so-called dry formaldehyde method of treating oats for smut is now used to some extent. This consists of sprinkling the oats lightly with a solution of 1 part formaldehyde (37 per cent to 40 per cent) to 1 part water, applied with a small hand sprayer or atomizer and covering for 4 hours. One quart of the solution will treat 50 bu. of oats. Seed potatoes were formerly treated with formaldehyde, 1 pint to 30 gals., for scab. It is not generally used for this purpose now, being superseded by corrosive sublimate. For disinfecting containers, storage quarters, etc., a solution of 1 pint formaldehyde to 10 gals. of water is recommended. As a disinfectant against certain soil organisms, formaldehyde may be used at a strength of 1 pint to 15 gals. of water, drenching the soil with 1 gal. of the solution to each cubic foot of soil. It is a good remedy for onion smut when applied to the soil in the drill rows at a strength of 1 gal. of formaldehyde to 50 gals. of water.

6. **Mercuric bichloride (corrosive sublimate).** — Corrosive sublimate is the most effective disinfectant for seed potatoes against *Rhizoctonia* and scab. The usual formula is:

Mercuric bichloride . . . . .	4 oz.
Water . . . . .	30 gals.

Corrosive sublimate is also used at a strength of 1 to 500 as a disinfectant for tools when cutting out fire-blight. It was formerly used on the wounds also in fighting this disease but proved to be ineffective on wounds and has been superseded by cyanide of mercury for that purpose (see below).

7. **Cyanide of mercury.** — Reimer (12) of the Southern Oregon Experiment Station in 1918 proved this chemical to be a much more effective disinfectant for wounds in cutting out fire-blight of pears than any other substances heretofore used. As a convenient disinfectant for both tools and wounds Reimer combined mercuric chloride and mercuric cyanide in the proportion of 1 gram of each in 500 cc. of water (see under control of fire-blight, p. 178).

8. **Organic-mercury compounds.** — During and since the World War a number of mercury compounds have been developed in Germany and placed on the market under such trade names as "Uspulun" and

"Germisan." These are said to have proved to be very effective seed disinfectants in extensive experiments carried out in Europe. More recently similar substances have been produced in America, such as "Semesan" and Corona mercury disinfectant. One of the advantages claimed for these new disinfectants is that they do not injure the seed in any way. It is even claimed that they increase the germinating powers of seeds in some cases. This is not true of formaldehyde and copper sulfate, the two fungicides most commonly used in the past. Some of the new fungicides appear to have some good qualities but it remains to be seen whether the new compounds or others to be developed will ultimately replace those formerly used for seed treatment.

9. **Creosote.** — One of the greatest economic problems in connection with the use of lumber is the protection of timbers against decay organisms. In case of timbers which come in contact with the ground, such as railroad ties, telephone and telegraph poles, pilings and fence posts, enormous losses result from the untimely decay caused by fungi. It has been found that the usefulness of such wood products can be prolonged many years by impregnation with creosote. This is now done on a large scale under pressure in large tanks, or by heating in open vats.

10. **Zinc chloride.** — Zinc chloride is sometimes used instead of creosote for impregnating timbers to preserve them from wood rots. It is a fairly good preservative where the wood is not exposed to severe weather conditions. Under extreme exposure the zinc will leach out more quickly than creosote and thus is not as effective as creosote for general outdoor use on railroad ties, bridge timbers, posts, etc.

11. **Sulfuric acid.** — Some success has been achieved with sulfuric acid as a soil disinfectant for damping-off fungi in forest nursery seed beds. A solution of 1 oz. of acid to 1 gal. of water is said to give good control.

12. **Calcium hypochlorite.** — This has been used with great success to sterilize seeds for use in physiological culture experiments (17).

**Heat as a fungicide.** — Heat can be used as a fungicide in disinfecting seed and in soil sterilization. As a seed disinfectant the use of heat has never become very wide-spread or effective. Its chief use has been confined to the hot water treatment for loose smut of wheat and loose smut of barley. On account of the very particular pains which must be taken in order to be successful with this treatment, it has never become very popular. More recently the hot water treatment has been used successfully for disinfecting bulbs infested with nematodes. As a soil sterilizer heat can be successfully used, especially on a comparatively small scale, such as in greenhouses or outside seed beds. For this purpose both hot water and steam have been used.

**Hot water.** — Soil sterilization with hot water consists essentially in drenching the soil with water hot enough to kill harmful organisms.

**Steam.** — There are several methods of applying steam to the soil to kill parasites (1, 6, 14).

*Steam-pan method.* — For this purpose galvanized iron pans of convenient size and 6 to 8 ins. deep are used. These are inverted and the edges forced a few inches into the soil. A steam connection is provided at one end so that the pan can be connected with a boiler or other source of steam. Steam is thus forced into the soil, which should be loose, and the pressure maintained long enough to sterilize the soil. Eighty to 150 lbs. pressure for 30 to 60 minutes should do the work.

*Perforated pipe method.* — This outfit consists of a number of perforated pipes connected with a source of steam. These pipes are placed about 18 ins. apart and 6 ins. deep. After the pipes are imbedded, the ground is covered with canvas and the steam is turned on.

*Steam rake or harrow method.* — This apparatus consists of a series of pipes with short sections coupled on like the teeth of a harrow. The "teeth" are hollow but pointed and have small openings near the tips for the escape of the steam. After the teeth have been forced into the ground it is covered with canvas or heavy cloth to keep in the heat and the steam is turned on.

*Tile method.* — This method (6) is a good one to install as a permanent system in the ground beds of a greenhouse. The system consists of laying tile lines about 30 ins. apart and about a foot beneath the surface. These tile lines are connected with the steam heating plant so that steam can be turned into them whenever needed.

**Purposes for which fungicides are used.** — As indicated in the outline near the beginning of this chapter there are two general objects to be attained in the application of fungicides, namely, (a) the protection of plants against infection, and (b) the disinfection of plants or of environmental factors such as the soil, pruning tools, containers, carriers or storage quarters. In the first case the process consists essentially in covering the healthy plant with some substance which will protect it against invasion by organisms which might come into contact with the plant subsequent to the application of the fungicide. Protection is essentially prevention rather than cure of disease. On the other hand, disinfection consists in killing the organism which is already present as spores, sclerotia, bacteria or in some other form.

**Protection.** — In the rôle of protective agents, fungicides may be applied (a) to the growing or dormant plant, (b) to wounds, and (c) to timber products as wood preservatives.

*Spraying and dusting.* — As a general rule the spraying or dusting of plants with various chemicals serves the purpose of protecting them from infection. There is a popular notion, more or less prevalent, that spraying is a cure for plant diseases; but in reality, except in rare cases, it is a preventive rather than a cure. Many of the preparations of copper and of sulfur such as bordeaux mixture, the lime-sulfur sprays and the copper-lime and sulfur-arsenate dusts are used for protective or preventive purposes. Spraying for apple-scab with lime-sulfur, for apple-tree anthracnose, peach leaf-curl and European canker with bordeaux, for late-blight of potatoes with bordeaux or copper-limedust, are all protective measures. The fact that such applications of sprays and dusts have a preventive purpose makes it absolutely necessary that these control measures be applied at the proper time. If the grower waits until infection occurs before spraying or dusting it is then too late to secure control by these practices.

*Wound dressings.* — This is a type of protection which should be used against certain diseases in which infection occurs in wounds on the trunk or branches of living trees. When pruning pear trees infected with fire-blight or when practicing surgery for the control of this disease it is necessary to apply a disinfectant to all cut surfaces to prevent reinfection by the blight bacteria which may be carried on the pruning tools. The wood-rotting fungi gain entrance through wounds. Infection by these organisms can be prevented by proper and timely disinfection of any wounds occurring in trees. There are two general types of wound disinfectants, (a) the air-porous type represented by bordeaux paste and cyanide of mercury, which protect because of their toxic effect, and (b) the air-tight type represented by coal tar, asphaltum and grafting wax, which protect by excluding all fungus spores or bacteria from the wounded surface.

*Wood preservatives.* — Manufactured lumber products which are exposed to the weather are subject to decays unless properly protected. Bridge timbers, pilings, railway ties, telephone and telegraph poles, and fence posts are examples of timbers which suffer most from wood-rotting organisms unless properly treated to prevent such damage. There are two ways of protecting timber from fungous decay, namely (a) by keeping it so dry that the fungi cannot grow, or (b) by the application of a toxic substance which will kill the fungi. Ordinary paints are helpful on lumber that is not in contact with the ground. They are not strongly fungicidal but act as more or less waterproof coverings. For timbers in contact with the ground impregnation with a toxic substance such as creosote or zinc chloride has given the best protection against decay.

**Disinfection.** — This term suggests the killing of a pathogenic organism already present. We have just learned that after an organism has once penetrated the host it is difficult to kill it with a fungicide. Disinfectants, for the most part, are applied to kill organisms carried on the surface of plants or plant parts, or on various elements of the environment where they may later come in contact with plants and thus infect them. Where the fungus or bacterium is present on the surface of propagative parts such as seeds, tubers, etc., it is often possible to disinfect these parts with something which will kill the parasite without injuring the seeds or other parts treated. The treatment of seed grain for smuts, potatoes for scab and *Rhizoctonia*, and of narcissus bulbs for nematodes comes under this heading. The last named is a case for hot water treatment rather than chemical, since the eelworms are inside the bulbs and cannot be reached by chemical disinfectants.

In some cases it is necessary to disinfect the environment. When the soil in greenhouses or seed beds becomes badly contaminated with damping-off fungi or nematodes a clean crop cannot be grown on such soil until it is disinfected. Both chemicals, and heat in the form of hot water or live steam, have been used for such purposes. When seed wheat is being treated for stinking smut the bags which have held the smutty grain should be disinfected before placing the treated seed back into them. Storage quarters which have held rotting fruits or vegetables should be disinfected when emptied before storing another crop. Tools which have been used in cutting out fire-blight should be disinfected after every cut is made.

As a rule where seeds or other plant parts carrying germs on their surfaces are to be disinfected, chemical fungicides such as formaldehyde, corrosive sublimate, copper sulfate or copper carbonate are used. If the organism is inside the host tissues, heat must be employed. For sterilizing containers, tools and soil either chemical fungicides or heat may be used. The choice will depend upon effectiveness, facility with which used, and availability. These will vary in different cases.

#### REVIEW QUESTIONS

1. On what two bases may fungicides be classified?
2. On the basis of nature, what are the chief two general types of fungicides?
3. What chemical elements form the basis for most of the common fungicidal sprays?
4. Describe the method of making home-made bordeaux mixture.
5. Describe the method of making ordinary commercial lime-sulfur.
6. Describe some of the sulfur sprays recently proposed as improvements over the common lime-sulfur.

7. What materials are in use in dust form as substitutes for bordeaux and lime-sulfur sprays?
8. For what two purposes in general are fungicides used?
9. Name some fungicides which have been most commonly used for disinfecting seeds.
10. Under what circumstances must heat be used instead of chemicals for seed-disinfection?
11. What substances may be used for protecting wounds on trees (a) against a bacterial disease such as fire-blight, (b) against infection by wood-rotting fungi?
12. Name some of the most effective preservatives for timbers which are exposed to the weather.
13. Name some good soil disinfectants for damping-off fungi.
14. What is the most effective way to use heat in disinfecting soil?

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## CHAPTER VIII

### DISEASE-FREE SEED AND NURSERY STOCK

Seeds and other parts of plants used for purposes of propagation, such as tubers, bulbs, cuttings and all sorts of nursery stock are capable of carrying the germs of disease from the old crop to the new or from one field or locality to another. In fact the great importance of this method of plant disease dissemination is not always fully appreciated. Barrus (2), Orton (20), Gardner (9), Chen (5) and others have discussed this means of transmission, and Orton and Beattie (21) have called attention to its relation to the plant quarantine problem. When the extensive dissemination of plant diseases through the use of contaminated seed and other propagating materials is fully realized it at once becomes evident that one very important feature of plant disease control is the use of disease-free seed and other propagating stock. In order to avoid the use of disease-carrying seed it is necessary to understand the various ways and the different forms in which disease-producing organisms may be seed-transmitted.

The causal agent of a disease may be carried either on the surface or imbedded within the tissues of the seed or other propagative part. The form in which the agent is carried depends in large part upon its nature, that is, whether it is fungus, bacterium, or virus. The following outline indicates the chief forms in which the various pathogenes or disease-producing agents may be carried.

#### I. Causal agent surface-borne.

##### A. On seeds.

##### 1. Fungi.

##### a. Spores.

##### 2. Bacteria.

##### B. On propagative parts other than seeds.

##### 1. Fungi.

##### a. Mycelium.

##### b. Sclerotia.

##### 2. Bacteria.

#### II. Causal agent borne internally.

##### A. In seeds.

##### 1. Fungi.

- a. Spores.
      - b. Mycelium.
    - 2. Bacteria.
    - 3. Virus.
    - 4. Nemas.
  - B. In other propagative parts.
    - 1. Fungi.
      - a. Spores.
      - b. Mycelium.
    - 2. Bacteria.
    - 3. Virus.
    - 4. Nemas.
- III. Neither in nor on seed but accompanying seed.
- A. With seed.
    - 1. Sclerotia.

### Surface-borne Pathogenes

While it is probably true that a majority of seed-borne pathogenes are carried internally, yet there are several notable examples of well-known and serious diseases the causal agents of which, in the form of fungus spores or sclerotia, or of bacteria, are carried externally on the seed or other propagative part.

**On seeds.** — There are both fungous and bacterial diseases in which the pathogene is carried over on the surface of seeds. Examples of fungous diseases perpetuated by means of spores clinging to the surface of seeds are certain smut diseases of the cereals. In case of stinking smut or bunt of wheat the spores of the causal fungus cling to the surface of the seed at threshing time and unless the seed grain is treated in some way to remove or kill the spores the new crop is likely to become smutted. The fungi causing the covered smut of barley and the oat smuts are transmitted in the same manner.

In case of the bacterial diseases it is more difficult to find outstanding examples in which the disease germs are carried on the surface of the seed. It is more often true that bacteria are borne internally, yet there are a few examples of externally-seed-borne bacterial diseases. The organism causing the tomato bacterial spot (*Bacterium vesicatorium* Doidge) is said to be carried on the surface of the tomato seed (10). In other cases, such as the bacterial blight of beans, the organism is found both externally and internally, although the germs borne within the seed are undoubtedly much more important in the perpetuation of

the organism. In Stewart's sweet corn disease the bacteria are carried largely on the surface of the seed although they also occur internally.

**On other propagative parts.** — One of the best examples of a fungous disease borne on the surface of vegetative propagative parts is the *Rhizoctonia* disease of potatoes. Here the fungus is perpetuated by means of sclerotia on the surface of the tubers. A closely related trouble, the violet root-rot disease, occurring on potatoes and other root crops such as the carrot is also perpetuated by means of surface-borne sclerotia. It seems probable that some of the powdery mildew diseases such as the apple powdery mildew, for example, may be disseminated in the form of mycelium on the twigs or buds of nursery stock. As an example of a bacterial disease the causal organism of which may be carried on the surface of propagative parts, the blackleg of potatoes may be mentioned. In this case the bacteria sometimes become smeared over the surface of healthy tubers through contact with decayed tubers. If such contaminated tubers are then used as seed stock without proper disinfection the disease may be transmitted to the progeny.

#### Causal Agent Borne Internally

The literature of plant pathology contains many examples of internally borne plant pathogenes. Among these are all types of parasitic diseases including those caused by fungi, bacteria and nematodes, as well as the virus diseases.

**In seeds.** — It was reported by Clinton (6, p. 292), that the fungus, *Phytophthora phaseoli*, which causes the downy mildew of lima beans is carried within the seeds as oöspores. It has been noted by several investigators, among them Bolley and Pritchard (3) and Hungerford (15, 16), that urediniospores and teliospores of some of the cereal rusts are sometimes found imbedded in the pericarp of the grain. Some investigators have advanced the theory that the rust fungus is perpetuated and transmitted from crop to crop by means of seed-borne urediniospores. Smith (23) believed that the grain rusts may be transmitted by means of dormant mycelium within the seed. Eriksson advanced the theory that the black stem rust of wheat is carried in the seed, not as either mycelium or spores but in a latent form which he termed "mycoplasma." However, Hungerford, by some extensive experimental work, has demonstrated beyond a reasonable doubt that in both the stem rust caused by *Puccinia graminis* (15) and the stripe rust caused by *Puccinia glumarum* (16), although spores do frequently occur imbedded in the grains, these rust fungi are not transmitted from crop to crop through the seed either as spores or mycelium or in any other form.

There are numerous cases in which disease-producing organisms are carried internally in seeds in the form of mycelium. The loose smuts (3) of wheat and barley are among the most frequently cited examples of this type. Here infection takes place at blossoming time and the mycelium enters the developing embryo and remains there as such throughout the period during which the seed is growing to maturity and also through the dormant period. It then continues development in the growing seedling, maturing its spores as the host plant reaches adult size. Bean anthracnose (24) is another good example of a disease in which the causal organism, *Colletotrichum lindemuthianum*, overwinters as mycelium and possibly also as spores within the tissues of the seed. *Phoma lingam* (14) causing blackleg of cabbage, and *Colletotrichum gossypii* (1), the cause of cotton anthracnose, are other examples of this type of seed parasitism.

In case of bacterial diseases of plants the bacteria themselves sometimes hibernate within seeds. *Bacterium phaseoli* (24), the cause of bean blight, is carried in this manner. Stewart's sweet corn disease is disseminated by bacteria carried within the seed corn, although in this case a large proportion of the organisms seem to be lodged on the surface of the grain, since seed disinfection affords a large measure of control, although it is not one hundred per cent efficient.

In the nematode disease of wheat caused by the eelworm, *Tylenchus tritici*, the larvae of the causal organism lie dormant in the diseased kernels until seeding time. If any seeds containing the nemas are left in the seed lot, when sown, the eelworms become active and resume their life-history development. Since diseased wheat grains are usually killed outright these organisms are not carried over in living seed, and perhaps, strictly speaking, this type of transmission should be listed under point III in the outline given above.

A few of the virus diseases of plants are now known to be seed transmitted. Bean mosaic (22) was shown to be transmitted in the seed as early as 1919. Seed transmission of clover mosaic was demonstrated in 1921 (7). It has recently been shown (8) that the virus of cucurbit mosaic is carried in wild cucumber seed but probably not in seed of the cultivated cucurbits.

**In other propagative parts.** — The causal organism of the powdery scab of potatoes, *Spongospora subterranea*, may be disseminated as spores in scabby tubers. *Phytophthora infestans*, *Verticillium albo-atrum*, and *Fusarium* spp. are carried in the form of mycelium in potato tubers. Fungi causing root-rot (*Armillaria mellea*) and such canker diseases as apple anthracnose (*Neofabraea malicorticis*), European canker (*Nectria galligena*), black-rot canker (*Physalospora Cydoniae*) and apple

blotch (*Phyllosticta solitaria*) may be carried as mycelium in nursery stock at the time of transplanting from the nursery. Such bacterial diseases as crown-gall and fire-blight frequently occur in the nursery. The causal bacteria are readily carried in stock when transplanted to the orchard. The bacterial organism causing potato blackleg is frequently carried in tubers as well as on the surface as cited above. Nemas or eelworms are carried in narcissus bulbs and in strawberry transplants. Examples of diseases transmitted by means of a virus carried in propagative parts other than seeds are the several virus diseases of potatoes carried in the tubers, the virus diseases of bramble fruits disseminated in the transplants and the mosaics of tomatoes and cucumbers which overwinter in certain perennial weeds.

#### **Causal Agent neither In nor On the Seed but Accompanying the Seed**

There are cases where the causal agent may be found along with the seed but not strictly attached to the seed either on the surface or internally. In case of ergot of grains and grasses the sclerotia may be mixed with the seed and unless removed before the seed is sown may be a source of infection later. The fungus causing stem rot of clover (*Sclerotinia trifoliorum*) is sometimes disseminated in the same way. Small sclerotia are dislodged from the clover stems at the time the seed is hulled and unless carefully removed may start the disease in the new crop. Probably the wheat galls produced by the nematode referred to above should be mentioned here since the blackened grains or galls containing the worms are not viable seed and may be likened, in a way, to the sclerotia just mentioned.

#### **Methods of Securing Clean Seed and Nursery Stock**

There are several different methods of obtaining clean seed, any one or all of which may be used in dealing with any particular disease, depending upon the nature of the disease, the host and other conditions. Some of these various means of control are: seed disinfection, selection, roguing, securing seed from a disease-free locality, the use of special seed plot, and the use of certified seed.

**Disinfection of seed and other propagative stock.** — The ease or difficulty of controlling plant diseases by this method depends to a great extent upon whether the causal agent is carried on the surface or imbedded within the tissues of the seed or other propagative part. Whenever a disease organism is borne entirely upon the surface of the seed material it is usually possible to get rid of it by careful application of

one of the various seed treatments which have been devised. Examples of this method are the familiar treatments of seed grain for smut by formalin, copper sulfate or copper carbonate, and the corrosive sublimate treatment of seed potatoes for *Rhizoctonia*. But if the causal agent is borne internally, then the problem of seed disinfection is much more difficult. The use of chemicals as disinfectants for this type of seed-borne disease is not feasible. Hot water has been used with some success as a disinfectant for loose smut of wheat and of barley where the parasite exists in the form of mycelium within the embryo of the seed. In this type of disinfection the vital question is concerned with the difference in the thermal death point of host and parasite. If this margin is so great that the seed can be heated sufficiently to kill the imbedded parasite without seriously injuring the vitality of the seed, then this type of disinfection can be used. Great skill and care are necessary, however, to use this type of seed treatment successfully.

**Selection.** — Wherever practicable clean seed or propagating stock should be selected. In some cases it may be possible to secure seed which is undoubtedly so clean that disinfection will be unnecessary. In other cases even careful selection may not insure clean seed. In the latter cases disinfection should be resorted to in addition to selection in order to make doubly sure of having seed as clean as it is humanly possible to make it. There are several methods which may be used in the selection of clean seed.

**Selecting clean seed from a mixed lot.** — It is sometimes possible, if feasible, to sort out clean seed from a lot containing some diseased seed. For example, one might select beans free from anthracnose in a field where the disease exists, or from a bin of potatoes one might select tubers which are apparently free from *Rhizoctonia* or scab. It is doubtful, however, if such selection could be 100 per cent efficient in most cases because of the difficulty of detecting all diseased seed. It is probable that some slightly infected seed would be overlooked. In case of the bean anthracnose, pod selection in the field might reasonably be expected to yield clean seed, but with potato *Rhizoctonia* it is impossible to insure clean seed by bin selection alone, therefore it would still be necessary to resort to seed disinfection in cases of this kind. When neither selection from a diseased lot nor disinfection prove effective, other methods are still available. Some of these means are mentioned below.

**Securing seed from a disease-free field or locality.** — It is often possible to find disease-free seed or propagating stock either in some field in the neighborhood or, if not, then in some other locality or section of the country. Shipping seed from a distance may prove

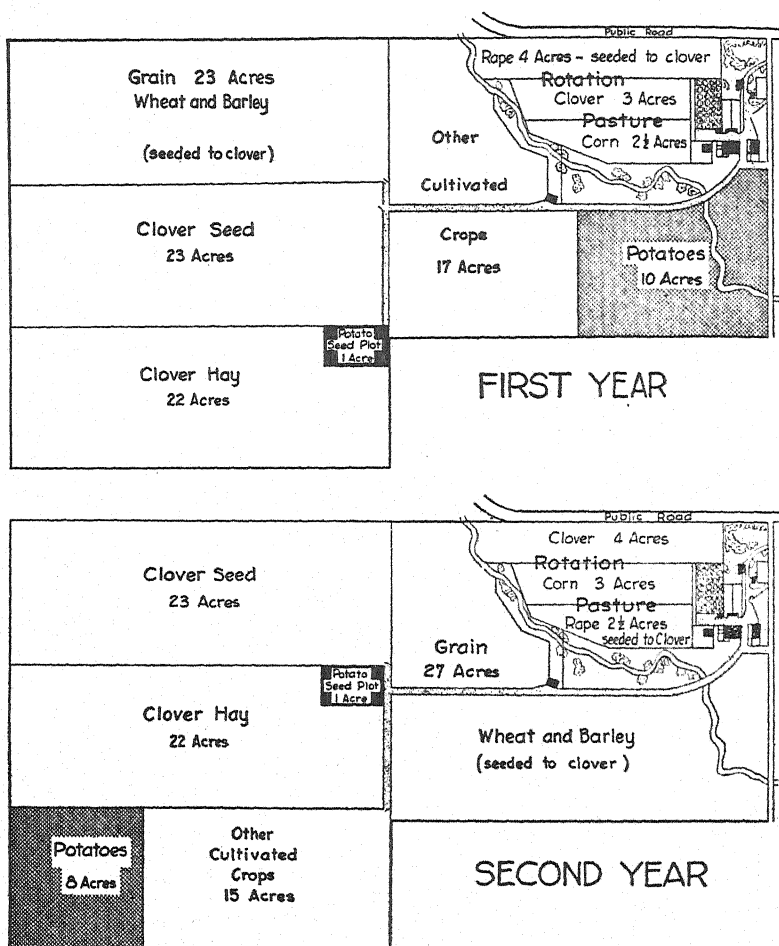


FIG. 9a. — (See also Figure 9b on next page.) Plan of a western Oregon farm showing suggested locations for the potato seed-plot and the main potato field each year in a four-year rotation for potato disease control. In these plans the seed-plot is separated from the main field of potatoes by at least 300 feet in order to guard against spread, from one field to another, of the mosaic and other virus diseases which are commonly insect-carried. The same general scheme could be adapted to other crops and other diseases. (After McKay, Ore. Agr. Exp. Sta. Bul. 221.)

expensive but it will usually pay in the long run if clean seed cannot be secured at home.

**Use of a special seed plot.** — In case of some diseases it is advisable to maintain an isolated plot for growing seed where a special effort is made to keep out diseases. The seed plot should be as far as possible

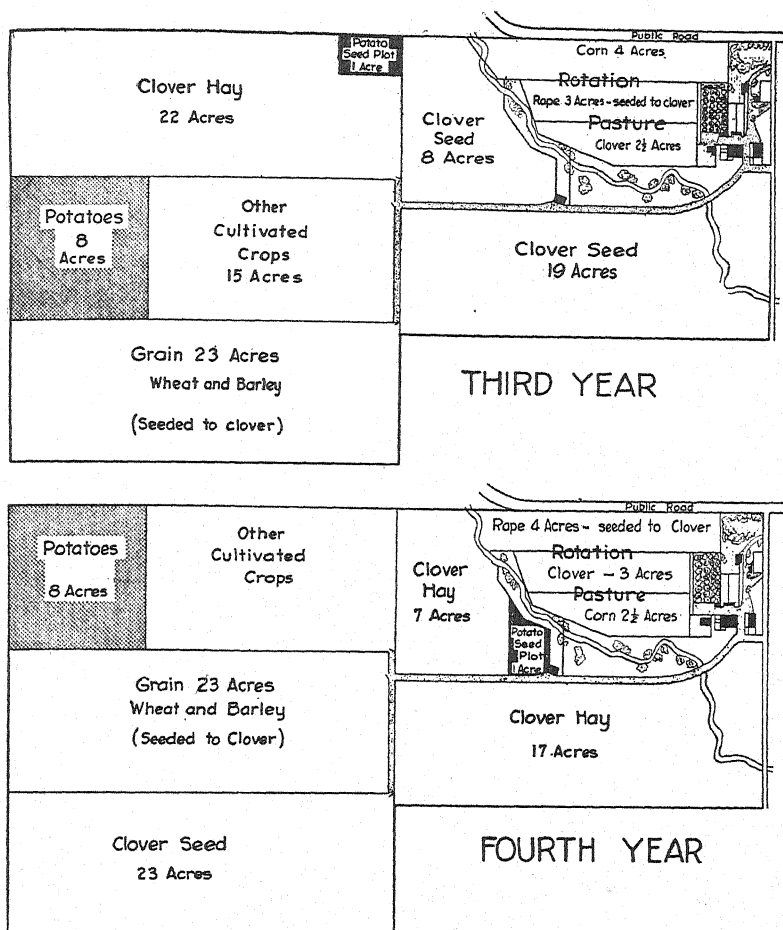


FIG. 9b. — For explanation see under Figure 9a. (After McKay, Ore. Agr. Exp. Sta. Bul. 221.)

from another field of the same crop and should be changed or rotated from year to year. The system of maintaining a special rotated seed plot applies especially to such potato diseases as *Fusarium* and *Verticillium* wilt where the causal organism persists for a longer or shorter time in the soil. For such diseases as potato mosaic the isolated plot is of especial importance because of transmission of the virus by aphids. Figure 9 shows a farm plan for rotating special seed plots.

**Roguing.** — With certain crops, such as potatoes, it is possible to detect many of the diseases during the growing season. The diseased



plants should be rogued or pulled out and destroyed as soon as detected. This practice is especially recommended in the special seed plot and is usually required where seed certification is practiced.

**Certified seed.** — During recent years a practice has sprung up in various states which promises much in the way of providing the grower with better seed, not only with reference to yield and quality but also in freedom from disease. The system of certification consists essentially in stated inspections by an authorized officer usually from the state agricultural college or experiment station and the issuing of a certificate to the grower which certifies as to the quality and freedom from disease of his product. The use of such certified seed by the grower should insure a better product in every way than the use of ordinary uncertified seed.

#### REVIEW QUESTIONS

1. In what ways may plant disease organisms be borne by seeds and other propagative stock?
2. In which case can seed be treated successfully by means of chemical fungicides, either liquid or dust?
3. In which case is it not possible to treat successfully with chemicals? What other treatment may sometimes be used with some success in such a case?
4. Discuss methods of obtaining disease-free seed and nursery stock.

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## CHAPTER IX

### QUARANTINE AND INSPECTION

The intelligent enactment and successful enforcement of any quarantine regulations necessarily presuppose a knowledge of various factors, among which are the nature of the parasite to be dealt with and of its host, and their inter-relations, together with any other factors entering into the introduction of a new disease. A necessary adjunct to the efficient operation of quarantine regulations is an adequate system of inspection. The following outline covers the chief points which should be kept in mind while considering this method of plant disease control.

#### I. Quarantine.

A. Reason for instituting quarantines.

B. Biological considerations.

1. The parasite.

- a. Nature of.
- b. Native home.
- c. Present distribution.
- d. Prevalence.
- e. Manner of spread.
- f. Climatic relations.

2. The host.

- a. Native home.
- b. Present distribution.
- c. Economic importance.
- d. Varietal susceptibility.
- e. Effect of breeding upon susceptibility.
- f. Close economic relatives that may be susceptible.

3. "Biological equilibrium."

- a. Of what it consists.
- b. Disturbance of the balance.
  - (1) Introduced parasites.
  - (2) Introduced hosts.

4. "Continuous" vs. "Discontinuous" spread.

- C. Geographical considerations.
  - 1. Contiguous *vs.* non-contiguous countries.
  - 2. Natural barriers.
    - a. Topography.
    - b. Climate.
- D. Immediate factors concerned in disease introduction.
  - 1. Agents.
    - a. Wind, insects, birds, etc. — not important.
    - b. Man most important agent.
  - 2. Medium on which introduced.
    - a. Plant products used for food and manufacture.
    - b. Living plants or plant parts used for propagation.
      - (1) Most important source of new diseases.
      - (2) Importing agents.
        - (a). Office of foreign seed and plant introduction.
        - (b). Commercial interests.
- E. Quarantine agents.
  - 1. Local or state governments.
  - 2. Federal governments.
- F. Quarantine laws.
  - 1. Federal.
  - 2. State.

## II. Inspection.

- A. As a factor in quarantine enforcement.
- B. Market inspection.
- C. Field inspection.
- D. Police power.

**Reason for instituting quarantines.** — Any particular plant disease or pest is usually not cosmopolitan in the beginning of its history, but has a more or less limited range in certain countries or regions. Sooner or later, however, due to the activities of man in transporting plants or plant parts from one country to another, this disease is likely to be carried to countries where it has hitherto not existed. When this happens, the introduced parasite sometimes attacks varieties of its host which have been introduced ahead of it, or closely related species which are indigenous to the new country, with much more virulence than it manifested in its native habitat. Instances are on record in which havoc has been wrought on a very important crop by invaders of this kind.

It is the danger of such economic losses from introduced parasites that has led governments to enact quarantine regulations in an endeavor to prevent the entrance of diseases and pests into territory not yet invaded.

**Biological considerations.** — In attempting to limit the spread of plant diseases by means of quarantines, a knowledge of the biology (5) of both parasite and host will be of great value.

*The parasite.* — In studying the parasite with a view to the necessity and feasibility of excluding it by means of quarantine, there are many things that must be considered. The nature of the parasite is important. Is it a slime mold, a bacterium or a fungus? What is its life history? How is it disseminated; especially is it carried on seed or other propagating stock? If so, is it carried externally or internally, as spores or mycelium, etc.? (See Chapter VIII.) Its native home and its present geographical distribution should be known. Its prevalence and virulence in its former habitat are of interest, although these do not necessarily indicate how serious it will be if introduced into new territory. The parasite should be studied carefully as to its environmental relations. Climate is a very important factor in determining the range of a disease and the probability of its becoming a menace if introduced into a new country.

*The host.* — The native home of the host, of course, was probably the same as that of the parasite. Its present distribution, if it is cultivated by man, may be quite different from the present distribution of the parasite, provided it was carried by man to new countries without taking its parasite along with it. The economic value of the host is of great importance. If the value is little or nothing, it may not be worth the trouble of a quarantine. On the other hand, if it is of great economic value, then a quarantine may be imperative in order to prevent enormous loss. The varietal susceptibility of the hosts should be known, especially if new varieties of the original host have been developed by breeding in the absence of the parasite. If close relatives of the host exist in the region to which the parasite is introduced, these relatives may prove to be less resistant than the original host. Therefore, in view of the above facts, a careful biological study of the host and closely related species and varieties should be made in connection with the establishment of a quarantine against any parasite liable to be introduced.

*"Biological equilibrium."* — In their discussion of the biological basis of foreign plant quarantine, Orton and Beattie (5) use the term "biological equilibrium" to indicate the condition that exists where a particular parasite has been living upon a host for so long that, due either to a reduction of toxicity on the part of the parasite or an increase

of resistance on the part of the host, or both, a state of affairs has come to pass in which the parasite is endemic, but seldom or never produces an epidemic; that is, the parasite, while always present on the host, is mildly parasitic and of relatively little economic importance. The attainment of this state of equilibrium is an evolutionary process and requires such a long time that if a parasite should be introduced into a region where congenial hosts are present, enormous damage, even to the extinction of the susceptible host, might occur before a state of biological balance would be reached.

Granting that in nature this state of equilibrium has been reached, there are several ways in which the balance may be disturbed as follows: (a) A parasite brought into a new country is able to attack a certain species native to this region with more virulence than it attacked its original host in its native habitat. An example of this is the chestnut-bark disease, caused by *Endothia parasitica*, which was introduced from Asia and has almost exterminated the native chestnuts in America. (b) Man's habit of carrying agricultural plants from one country to another has introduced hosts to regions where new parasites attack them as in case of the downy mildew, caused by *Sclerospora philippinensis* Weston, which attacks maize, an introduced plant, in the Philippine Islands. (c) Another phase of disturbed balance brought about by introduced hosts is illustrated by the asparagus plant and its rust, caused by *Puccinia asparagi* D. C. In this case the host was brought to America from Europe without its parasitic rust. After long cultivation and the development of new varieties in this country, the rust was finally introduced and threatened to destroy asparagus culture here until new strains of the host plant were brought in from Europe where the state of equilibrium had long existed and resistant strains of asparagus were to be found.

"Continuous" vs. "Discontinuous" spread. — Butler (1) has used the terms "continuous spread" and "discontinuous spread" to apply to the more or less slow and gradual natural spread within a given region, and to the long jump from one country or zone to another, respectively. An example of the former type of spread is the chestnut blight fungus, which has spread by natural means from the point or points of original introduction into this country until it has almost reached the limits of distribution of its host. A phase of continuous spread is illustrated by fire-blight which, according to the statement of Gossard and Walton (3), may spread from south to north each spring following the seasonal blossoming of the fruit trees which it attacks. The discontinuous type of spread is illustrated by the white pine blister rust fungus, *Cronartium ribicola* F. de Wal., which was introduced from

Europe into North America by making the long jump across the Atlantic Ocean, and also by the original introduction of the chestnut blight from Asia to America.

Evidently the discontinuous type of spread is the more readily amenable to control by quarantine methods because diseases which are spread in this manner are nearly always carried by man on plants or plant parts. It is true that diseases scattered by the continuous type of spread are sometimes also carried by man, but in this type there is always the spread by natural agents which quarantine cannot reach.

**Geographical considerations.** — It is obvious that the spread of plant diseases between **contiguous** countries which offer a similar environment is just as likely to occur by the continuous type of spread as is the distribution of diseases within a country, since political boundaries between such countries offer no obstacle to such spread. Between **non-contiguous** countries, however, the spread is necessarily of the discontinuous type, unless the two countries happen to be on the same continent, offer a similar environment, and are separated only by another country through which continuous spread may take place. Plant quarantines are apt to be more effective between non-contiguous countries, although they are frequently established between contiguous countries or even between parts of the same country. Diseases like potato wart which depend almost wholly upon man's operations for dissemination can be effectively restricted by rigorous quarantine measures. The potato wart disease has spread but little beyond the limits of the small area in Pennsylvania where it was first discovered in this country. Even a disease such as white pine blister-rust which spreads readily in nature, may be retarded in its spread by quarantine between contiguous countries or parts of the same country because of the fact that it is disseminated extensively on propagating stock shipped from place to place.

The **natural barriers** which may intervene to prevent the spread of plant diseases are of two kinds, topographic and climatic. The chief topographic factors are oceans, deserts and mountain ranges. Large bodies of water are undoubtedly effective barriers, and mountains may be so, but definite information on the latter is not at hand. Climate and weather play a very important part in the life of parasites (see Chapter V), and probably climatic factors act as barriers in many cases.

An interesting but baffling case is that of the stripe rust fungus, *Puccinia glumarum* (Schmidt) Erik. & Henn. This fungus has been known in the western United States since 1915 and there are evidences that it has been present in the Pacific coast region for a much longer time. To date this rust has never been reported east of a line running

north and south along the eastern foothills of the Rocky Mountains, and the Black Hill region of South Dakota. Why this disease has never spread farther eastward into the great wheat region of the Middle West is a difficult question. Perhaps the great plains region offers just the right climatic or topographic conditions or a combination of these to act as a barrier. Possibly there is some other factor concerned in this case.

**Immediate factors concerned in disease introduction.** — The immediate agents concerned in the introduction of a new disease are of two general types, namely, those which act in nature such as wind, birds, insects, etc.; and man. While in nature some diseases are disseminated by wind, insects, etc., this method of spread is mostly of the continuous type and these agents are probably not of great importance in the long-jump type of spread. In the latter, man is undoubtedly the most important agent.

The medium on which pathogens are introduced is nearly always some plant or plant part. This medium may be considered under two general heads: (a) plant products used for food or manufacture, and (b) living plants or plant parts used for propagation. The former is of little danger unless the products are diverted from their original destination and used for propagation. The latter is the chief source of all our introduced diseases.

The chief importing agents of propagating stock are (a) The Office of Foreign Seed and Plant Introduction of the United States Department of Agriculture (6), and (b) commercial interests. The former agency takes such extreme precautions with all the stock imported under its direction that there is little or no likelihood of bringing in any dangerous disease on such importations. Practically all of the serious diseases thus far brought into this or any other country have come in on propagating stock imported by commercial interests. It is chiefly by reason of such traffic that quarantines have become a necessity as one important agency in the control of plant diseases.

**Quarantine agents and laws.** — The agents endowed with authority to institute quarantines are the State and Federal Governments. The former makes laws to prevent the introduction of diseases and pests from outside sources into its own territory or from one part of the state into another part. The Federal Government enacts quarantine legislation to guard against the introduction of diseases from foreign countries. It can also promulgate interstate quarantine regulations. To meet the need for exclusion of plant diseases which might be introduced from foreign countries into this country the United States Government enacted the Federal Quarantine Act of 1912 and subsequent amend-



ments. This act gives the Secretary of Agriculture power to promulgate orders forbidding the importation into the United States of any plants, plant parts, or plant products which he shall determine necessary in order to prevent the introduction into this country of any dangerous plant disease or pest. However, provision is made for the importation of plants or plant parts by the Department of Agriculture for experimental or scientific purposes under such regulations as may be prescribed to guard against bringing in diseases or pests. Interstate quarantine against plant diseases or insect pests is provided for under acts of August 20, 1912, and March 4, 1917, which stipulate that whenever a plant disease or pest is introduced into or exists in a limited area of the United States, the Secretary of Agriculture is authorized and directed to promulgate such quarantine and inspection regulations as seem necessary to prevent the spread of such plant diseases into uninfested territory. The various states also have passed quarantine and inspection laws modeled largely after the Federal acts.

**Inspection.** — Inspection of plants and plant parts for the presence of diseases or pests has become one of the extensive and important phases of control. There are three types or phases of inspection, each of which fills a particular need and is of great importance if the campaign against plant diseases and pests is to be made effective. These three types of inspection, or perhaps we should say, three different fields in which inspection has been instituted, are (a) as an aid in enforcing quarantine regulations, (b) market inspection, and (c) field inspection as an encouragement to the practice of better control methods.

*Inspection a factor in quarantine enforcement.* — As a factor in quarantine enforcement, a system of inspection has been developed whereby an attempt is made to detect and detain shipments of plants or plant parts which carry dangerous diseases while at the same time permitting the entrance of clean stock. The degree to which this inspection can be made efficient will determine the extent to which the importation of propagating stock can be permitted without too great danger of bringing in serious pests. There are, however, certain things which tend to reduce the effectiveness of this type of inspection. These are the facts that some parasites are carried internally and are impossible of detection with the means available to the inspector; and that even though some diseases show external evidences of their presence, they may not be very prominent and may escape the inspector's notice, since the human powers of observation are not usually 100 per cent efficient.

*Market inspection.* — Government inspectors are now stationed in all large cities. It is their duty to inspect agricultural produce, especially fruits and vegetables, with the purpose of eliminating, as far as possible,

the enormous losses that occur in perishable products due to disease and decay. This phase of plant pathology is discussed in detail in Chapter XIII.

*Field inspection.* — An important type of inspection service is that represented by the county, district, or state horticultural inspectors, among whose duties is the inspection of private premises, especially orchards and other fruit plantations, with the view of lessening the amount of disease through the encouragement of better and more rigidly enforced control measures.

*Police power.* — The duties involved in the various types of inspection may sometimes be combined in one person. For example, a county horticultural inspector may be called upon to act in case of quarantined propagative stock imported into his territory, to inspect nursery stock grown in his county, or to inspect orchards for evidence of negligence on the part of the owner in applying proper control measures. All of these duties require that the inspector be invested with police authority to enforce his rulings. The wielding of this power calls for tact and good judgment on the part of the inspector. Thus a good inspector must be both a good plant pathologist and a diplomat. It is inevitable that misunderstanding and conflict should arise between Governmental authority and private interests when restrictions are imposed by quarantine regulations. However, strong pleas (4, 7) have been made for moderation on the part of quarantine authorities and for coöperation between private and governmental interests, so that it seems likely that, as time goes on and better understanding is arrived at by all concerned, adjustments will take place whereby diseases may be successfully excluded without imposing any undue hardships upon commercial interests.

#### REVIEW QUESTIONS

1. Why are quarantines sometimes advisable?
2. What biological considerations must be taken into account in determining whether or not a quarantine is advisable?
3. In what sense is the term "biological equilibrium" used?
4. Discuss "continuous" versus "discontinuous" spread of disease in relation to the establishment of a quarantine.
5. What barriers are effective in preventing the natural continuous spread of disease?
6. What agencies are chiefly responsible for the long-distance introduction of a disease into a country where it has hitherto not existed?
7. On what media are new diseases usually brought in?
8. Are introduced diseases always as virulent on their new hosts as in their original habitat?
9. What agencies are endowed with power to promulgate quarantines?
10. What machinery is necessary for the enforcement of quarantines?

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## CHAPTER X

### "SICK SOIL"

The term "sick soil" as here used is applied to the condition which sometimes occurs when a particular area of land will not produce a satisfactory yield of a certain crop or crops. This condition may be due to any one or more of several factors. When this "sick" condition pertains especially to a particular crop the soil is sometimes said to be sick for this crop and such terms as "wheat-sick soil," "flax-sick soil," etc., are used to imply that this soil will not produce a good yield of this particular crop. The soil conditions, both normal and "sick," as here discussed are indicated in the following outline:

- I. The normal soil.
  - A. Physical and chemical properties.
  - B. Biological properties.
    1. Beneficial soil flora.
      - a. Bacteria and fungi.
- II. Sick soil.
  - A. Due to non-pathogenic factors.
  - B. Due to plant pathogenes.
    1. Parasitic soil fungi.
      - a. Damping-off fungi.
      - b. Wilt fungi.
      - c. Root-rot fungi.
      - d. Miscellaneous.
    2. Parasitic soil bacteria.
    3. Parasitic animal life.
  - C. Control.
    1. Soil sterilization.
    2. Crop rotation.
    3. Changing soil reaction.
    4. Use of clean seed.
    5. Use of resistant varieties.

#### The Normal Soil

Before entering upon a discussion of the factors involved in causing sick soils, let us consider briefly, by way of comparison, those conditions existing in what is considered as the normal, healthy soil. Of course

the physical and chemical make-up of the soil play an important part in soil fertility and productiveness, but at this point we wish particularly to call attention to the beneficial microorganisms which are indispensable to modern agriculture as contrasted with the harmful organisms which are concerned in sick soil conditions. The soil flora consists chiefly of bacteria, fungi and algae. The soil also contains many minute forms of animal life such as protozoa, nemas and other minute forms which should be mentioned in a discussion of this subject. The bacteria and fungi are probably of much more importance than the other forms and deserve particular mention in this connection.

**Bacteria.** — Students of botany are at least somewhat familiar with the part played by bacteria in the decay of organic matter in the soil, and especially with their rôle in the nitrogen cycle of higher plants. Bacteria, as well as the molds, are concerned with the decomposition of cellulose and with the cleavage and fermentation of sugars, starches and gums. They probably also help in the decomposition of fats and waxes found in vegetable humus. One of the most important functions of bacteria in the soil is the transformation of protein substances, in several consecutive steps, into ammonia, nitrites and nitrates.

*Ammonification.* — Certain species of bacteria, among them *Bacillus mycoides*, are able to secrete enzymes which aid in breaking proteins into various cleavage products such as peptones, amino-acids, carbon dioxide and ammonia.

*Nitrification.* — It has been established that certain other bacteria are able to oxidize ammonia to nitrites. These bacteria are designated nitrous or nitrite bacteria. Still other forms known as nitric or nitrate bacteria have the ability to carry the oxidation a step further and form nitrates, which are immediately available as raw food materials for plant growth. Recently evidence has been brought forth to show that some plants can use ammonia directly thus eliminating the necessity for these further steps in nitrification.

*Nitrogen-fixation.* — There are two types of bacteria which are able to fix atmospheric nitrogen in a form available as plant-food material. Certain forms are *symbiotic*, as the well-known nodule-forming species on leguminous plants. Other species are known to live symbiotically on non-leguminous plants. Some *non-symbiotic* species, both aërobic and anaërobic, are also able to fix free nitrogen.

**Fungi.** — Certain molds are able to cause an accumulation of ammonia in the soil by the decomposition of organic matter in much the same manner as the ammonifying bacteria. Fungi also are probably more important than bacteria in the decomposition of cellulose in the soil.

In addition to the beneficial fungi of decay, there are fungi which are

associated with the roots of living plants. This relationship is known as *mycorhiza*. There are two general types of mycorhiza, the *ectotrophic* type in which the fungus hyphae attach themselves to the surface of roots or live inter-cellularly, and the *endotrophic* type in which the hyphae are intra-cellular in the root tissues. It has been claimed that many of these mycorhizal relationships are beneficial and even necessary to the life of the higher plant. Some forest trees, it is said, will not thrive in soil devoid of the special fungi which live in this relationship upon their roots. On the other hand it has been claimed (18) that many of the fungi associated with the roots of trees are parasitic rather than symbiotic as has been supposed.

### Sick Soil

There are many different factors concerned in bringing about an abnormal, unhealthy, or "sick" condition in soils. If any of the beneficial soil organisms mentioned above should fail to function properly, it might bring about a condition in which crop plants would fail to grow and produce as desired. But in addition to the failure of these organisms to perform their proper function there are other unfavorable conditions which may give as much or even more trouble. These factors may be grouped under two heads, namely, (a) sick soils due to non-pathogenic factors, and (b) those due to harmful parasites.

**Non-pathogenic factors.** — Acid and alkaline soils have been blamed for crop failures but it is probable that these conditions, except in extreme cases, do not act directly upon the crop plants but rather indirectly through their influence upon the various soil microorganisms. This influence may be exerted upon either the beneficial organisms previously mentioned or the harmful organisms discussed below. Of course there are extreme cases of alkali soils, nitre-sick soils, etc., where there is undoubtedly a direct toxic or other harmful action upon crop plants due to excess of these soil constituents.

A common cause of soil trouble is the presence of too much cellulose-containing material in the soil. It is a matter of common observation that the addition of straw to the soil cuts down the yield the first year. It is now known that the organisms which bring about the early stages of decay in cellulose draw heavily upon the available supply of nitrogen in the soil and thus compete with the crop plants to the great detriment of the latter (1, 6, 19). The common practice of burning the straw in wheat farming sections overcomes this immediate effect but is a harmful practice in the long run because the humus content of the soil is not properly built up and conserved.

**Plant pathogenes.** — While the plant pathologist has to be cognizant of the various factors governing soil productivity discussed above, these phases of the subject are not his special field. The study of the group of soil bacteria concerned with ammonification, nitrification, nitrogen-fixation, etc., belongs especially to the bacteriologist, and the study of the other non-pathogenic influences falls either in the field of the soil bacteriologists or in the field of other soil specialists. While all of these phases of the subject may overlap to some extent, the particular phase of the problem which falls more especially within the field of plant pathology has to do with the investigation of the pathogenic organisms found in the soil. There is no doubt that a great deal of soil sickness which was formerly attributed to various other causes is really due to specific parasites which live for a greater or less length of time in the soil, especially on plant debris, and render such infested soil incapable of producing satisfactorily the crops which are susceptible to these specific organisms. Bolley (3) was one of the first to call attention forcibly to such "sick soil" conditions and point out the relation of root parasites to this type of crop failure.

**Damping-off fungi.** — A serious trouble often encountered, especially in seed beds, in greenhouses or under other circumstances where large numbers of plants are grown under crowded conditions with high humidity and poor ventilation, is a sudden rotting off and lopping over of seedlings, usually known as damping-off. There are several different fungi which may cause damping-off. Some of the most common are species of *Pythium*, *Corticium* and *Fusarium*. Some other genera which have sometimes caused damping-off are *Botrytis*, *Trichoderma*, *Pestalotzia*, *Sclerotinia* and *Phytophthora*.

One of the most prevalent causes of damping-off and one of the earliest studied is *Pythium debaryanum*. This fungus is frequently met with in greenhouses and seed beds of various kinds, not only in gardening sections, but frequently in forest nurseries. For more detailed discussion of this fungus see page 225 in Part II. The *Rhizoctonia* fungus, *Corticium vagum*, is a serious cause of damping-off in seedlings as well as a cause of root-rot and other troubles in various plants. (See Potato *Rhizoctonia* in Part II.) The genus *Fusarium* contains an enormous number of species, some of which cause damping-off and a large number of which cause wilts, rots, and other troubles mentioned below.

**Wilt fungi.** — The genera *Fusarium* and *Verticillium* contain some of the most common species of wilt-producing fungi. *Rhizoctonia* sometimes also produces a wilting in addition to other symptoms. *Fusarium oxysporum* produces a well known potato wilt, *F. lini* causes wilting of flax, and *F. nivium* is the cause of wilt in watermelons. Cabbage yellows

is due to *F. conglutinans*, a wilt of tomatoes is caused by *F. lycopersici*, and spinach wilt is due to *F. spinaciae*. Plant pathological literature contains references to many other wilt diseases caused by species of *Fusarium*. *Verticillium alboatrum* is the cause of a wilt disease of potatoes which in certain regions outranks the *Fusarium* wilt of potato in economic importance. This same species of *Verticillium* also causes a wilt of egg-plant, cucurbits and other plants.

**Root-rot fungi.** — There are many species of fungi which have the ability to cause root-rot in plants. Among the genera containing root-rotting species are *Helminthosporium*, *Fusarium*, *Rhizoctonia*, *Corticium*, *Ophiobolus*, *Gibberella*, *Thielavia*, *Sclerotinia* and *Armillaria*.

Bolley (3), in 1913, stated that in North Dakota wheat sickness had become a common condition of the soils in that great wheat-growing region. He claimed that the falling-off in quantity and quality of the wheat produced there was due more to wheat parasites than to a depletion of soil fertility due to constant cropping to wheat as was the general opinion at that time. Among the fungi which he claimed were responsible for this wheat-sick condition are species of *Fusarium*, *Helminthosporium*, *Alternaria*, *Macrosporium*, *Colletotrichum* and *Cephalothecium*. Probably not all of the fungi isolated by Bolley are strongly parasitic on wheat roots, but some of them, especially *Fusarium* and *Helminthosporium*, have been shown by other and later investigations to be all that Bolley claimed for them.

Henry (10) has recently shown that *Helminthosporium sativum*, *Fusarium graminearum* (*Gibberella saubinetii*) and *F. moniliforme* are among the most virulently pathogenic organisms attacking the roots of wheat. During the last ten years a number of papers have been written on various phases of the root-rot problem in wheat and corn (2, 11, 12, 13, 14, 15, 16, 24). The Take-all disease of wheat is caused by *Ophiobolus cariceti* (14) which works mainly in the roots. The dry root-rot of beans is due to *Fusarium martii phaseoli* (5). A serious root-rot of peas is caused by *Aphanomyces euteiches* Drechsler.

The *Rhizoctonia* fungus, *Corticium vagum*, is responsible for a great deal of root-rot trouble among cultivated plants. Peltier (20) has reported that *Rhizoctonia* attacks a large number of truck crops, among them the following: beet, bean, cabbage, cauliflower, celery, cucumber, eggplant, horseradish, lettuce, muskmelon, pepper, radish, squash, sweet potato, pea, parsnip, potato and tomato. The violet root-rot of potato, onion, asparagus, carrots, clover and many other plants is due to *Rhizoctonia crocorum*. A root-rot of beans, horseradish, tobacco and watermelon is caused by *Thielavia basicola*. Species of *Sclerotinia* cause root-rots in such plants as clover and carrots.



*Armillaria mellea* causes the well-known root-rot of both fruit trees and forest trees.

This list is by no means complete, but the large number cited serves to show the extreme importance of the root-rot fungi as a cause of sick soils.

**Miscellaneous soil fungi.** — In addition to those soil fungi which may be designated as damping-off, wilt-producing or root-rotting fungi, there are others of a miscellaneous nature which persist in the soil for a longer or shorter period of time, and which should be mentioned here. Examples of some of these fungi are the onion smut fungus, *Urocystis cepulae*, the cabbage club-root organism, *Plasmidiophora brassicae* and the potato wart parasite, *Synchytrium endobioticum* (see description of club-root in Part II).

**Parasitic soil bacteria.** — Apparently there are not as many parasitic bacteria as there are fungi which live indefinitely in the soil, yet there are a number of bacterial diseases of plants, the causal organisms of which can live for some time in the soil. Among these may be mentioned the bacterial brown rot of potatoes, tomatoes and other plants caused by *Bacterium solanacearum*, the soft rot of vegetables due to *Bacillus carotovorus*, and the crown-gall organism, *Bacterium tumefaciens*.

**Nematodes.** — The eelworms or nemas are the source of a great deal of soil sickness. The most important types of eelworm diseases are the root-rot caused by *Heterodera radiculicola*, the leaf, stem and bulb trouble due to *Tylenchus dipsaci*, the wheat-gall disease caused by *Tylenchus tritici* and the sugar-beet disease due to *Heterodera schachtii*.

**Control.** — There are several methods of control especially adapted to sick soil troubles due to parasitic organisms. Some of the more important of these are soil sterilization, crop rotation, changing the soil reaction, the use of clean seed and the introduction of resistant varieties of crop plants.

**Soil sterilization.** — This method consists in using chemicals or heat to destroy completely all parasitic organisms in the soil. It can be used advantageously in the greenhouse and in seed beds but has practical limitations in dealing with field crops. This method is discussed briefly in Chapter VII.

**Crop rotation.** — The usefulness of this method of eliminating sick soil conditions rests upon the fact that many of these deleterious organisms will gradually die out if the susceptible crop is not grown on the infested soil for a number of years. The most important thing to know in this connection is the length of time an organism will live in the soil in the absence of its host crop. Crop rotation is a fundamental agricultural practice but the presence of a long-lived sick soil organism

in a field may necessitate a longer rotation cycle than would otherwise be used.

*Changing the soil reaction.* — There are some cases in which the growth of a particular organism is favored by certain chemical conditions of the soil and hindered or inhibited by other conditions. For example, the cabbage club-root organism thrives better in a somewhat acid soil than it does in an alkaline soil. In this case it is possible, by adding lime, to correct the acidity of the soil so that it is rendered unsuitable for the growth of the club-root parasite, without affecting the host adversely.

*Clean seed.* — Many of the organisms involved in sick soil conditions are already present as native inhabitants of the virgin soil. Other sick soil organisms are not native but are introduced in one way or another, often many years after the soil has been put under cultivation. The chief way in which such organisms are introduced is on diseased seed. Careful selection or treatment of seed liable to carry these disease-producing organisms will oftentimes prevent the introduction of serious sick soil diseases.

*Resistant varieties.* — This, of course, is a method of combating disease which is to be recommended wherever feasible. One of the best examples of the use of this method in fighting sick soil diseases is that of the development of yellows-resistant varieties of cabbage in Wisconsin during recent years.

In addition to the above-mentioned practices, there are certain other methods which may be an aid in particular instances. These will be discussed in connection with any of these soil-infesting diseases that are taken up in detail in Part II.

## REVIEW QUESTIONS

1. Mention the factors which make a soil normal for ordinary plant growth.
2. Mention the chief types of beneficial soil flora and explain how and why they are beneficial.
3. What two general types of factors may make a soil "sick"?
4. Mention several types or groups of fungi which are responsible for sick soil conditions.
5. What is meant by such terms as "wheat-sick soil," "flax-sick soil," "cabbage-sick soil"?
6. When a certain soil fails longer to produce a crop of wheat or any other cultivated crop, does that fact necessarily mean that the fertility of the soil is exhausted?
7. Name some fungi which may make the soil sick for wheat; for flax; for cabbage; for coniferous forest-tree seedlings.
8. Discuss control measures for sick soil conditions.

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## CHAPTER XI

### DISEASE RESISTANCE AND IMMUNITY IN PLANTS

The selection and breeding of plants resistant or immune to disease affords one of the most profitable and promising lines of work in the whole field of plant-disease control. Such methods of controlling diseases as spraying, seed disinfection, surgery and all the other methods discussed in previous chapters are expensive and sometimes inefficient means of combating plant diseases. All these various methods are mere expedients to which we must resort because, at present, we have no better way of contending with the various maladies which take toll of our crops. If we could find or develop varieties of plants immune to all diseases and at the same time possessing all the commercially desirable qualities, we could dispense with spraying and the other expensive and profit-eating control practices which are now necessary in order to grow crops reasonably free from disease. However, the task of securing resistant or immune varieties of crop plants is a long and arduous one and we cannot hope to be rid immediately and entirely of the necessity for using many of the expedients just mentioned. While many notable successes have already been achieved by plant pathologists and plant breeders in the development of resistant and immune varieties, an enormous amount of work remains to be done before we can begin even to approach the final goal. Some of the items which are of interest in a discussion of this topic are: (a) the advantages of the use of resistant and immune varieties over other methods of disease control; (b) the basis of resistance and immunity in plants; (c) disease-resistant *versus* disease-escaping plants; (d) the means of securing resistant or immune varieties; (e) the genetics of resistance and immunity; and (f) the difficulties met in developing resistant varieties.

**Advantages of Immunity.**—There are several reasons why it is desirable to find resistant varieties of plants which can be substituted for the more susceptible ones. (a) In some cases it has never been possible to secure adequate and satisfactory control by any of the ordinary practices in use. In such cases the development of resistant varieties makes control possible where it was previously impossible or only partially and unsatisfactorily accomplished by the other means available. The fire-blight of pears and other pome fruits is a good example of this type of disease. Heretofore this disease has been fought largely by the expedient of cutting out diseased parts, a process which is

expensive and one which can never be guaranteed completely to eradicate the disease. Recently, resistant pear stocks have been discovered which, while not bearing desirable fruits, nevertheless can be used for root and trunk framework on which to top-work desirable varieties. This gives a tree which can never be killed outright by the blight. It is probably only a matter of time until fruit qualities will be improved to such an extent that even the fruit-bearing branches may also be immune to blight. The black stem rust of wheat is another example of a disease which cannot be satisfactorily controlled by any of the other methods. Attempts to control it by removing the alternate host, the barberry, have been partially successful but probably will never be entirely effective because of the difficulty of finding all barberry plants, especially where they have escaped from cultivation, and also because of the probability of uredospores being carried long distances by winds. In view of these facts the development of resistant varieties of wheat seems to be the most promising method of controlling this rust. (b) In other cases where control is possible by one or more of the methods other than the use of resistant varieties, the cost of control or other difficulties may be such that attempts at prevention by these means are not always justified. Growers sometimes do not feel that they can go to the expense of an elaborate spray schedule for certain fruit diseases which do not occur every year but are prevalent only at irregular intervals which are unpredictable in advance. They feel that they can take a chance on sustaining considerable loss once in several years rather than meet the certain expense of a regular control program each year. If immune varieties were available the occasional loss could be eliminated without spending the large amounts each year for protection. In other words they could eliminate the yearly premium which they have to pay for insurance against losses. (c) In still other instances where certain measures may be effective in controlling the disease, the treatment causes injury to the host which offsets the gain derived from disease control. Certain seed treatments for the cereal smuts cause serious injury to the seed, and some spray materials injure the fruit and foliage to such an extent that it is questionable whether the remedy is not worse than the disease. (d) Even in those cases where disease control by other methods is both possible and profitable, if immune varieties of suitable quality were available the profits might be increased by the elimination of the expense of other control measures. Thus from every point of view the development and use of resistant or immune varieties of crop plants is highly desirable.

**Nature of disease resistance.** — Perhaps no phase of plant pathology has received more attention or has been more difficult of solution than

the question as to the basis of resistance and susceptibility to disease in plants. Much work has been done and many lines of investigation have been carried out more or less thoroughly, but the problem is yet far from ultimate solution. There are so many different degrees of resistance, real or apparent, that it seems evident that no one factor is responsible in all cases. For convenience in discussion, all disease resistance in plants may be considered under two heads, (a) apparent resistance, and (b) true resistance. The first of these may be interpreted to include all cases in which, for some incidental reason, environmental or otherwise, plants which normally would be expected to become diseased escape or avoid disease. The second class includes all cases in which resistance is due to some characteristic, physiological or structural, inherent in the plant itself. This classification is only tentative and no hard and fast line can be drawn between the two classes; first, because of lack of sufficient knowledge of all the factors involved; and secondly, because some factors, as environment, may function in the incidental avoidance of disease, or on the other hand may be involved in the production of truly resistant characteristics.

**The basis of true resistance.** — Enough work has now been done on this problem to enable us to say that in all probability the basis of resistance is not the same in all cases but that a considerable number of different factors may be concerned. As the work has progressed it has become more and more evident that generalizations cannot be made indiscriminately but that every case has to be considered on its own merits. Thus it seems that the probable basis of resistance in wheat to stem rust is not the same as the factor which is the basis of resistance to onion smudge in certain varieties of onions. In general all factors responsible for true resistance may be listed under two heads, (A) those involving structural characteristics of the plant, either external or internal, and (B) those involving the physiological reactions of the host cells. Specifically, a number of different factors, each in connection with certain specific diseases, have been named by various workers as the basis of resistance to disease in plants. Some of these suggested factors include: (a) external structure, such as stomata, cuticle, hairs, waxes, etc.; (b) internal structure, especially with reference to cell walls and particularly the middle lamella; (c) cell-sap acidity; (d) tannin; (e) "antagonism" between the physiological reactions of host and parasite; (f) availability of food for the parasite; (g) osmotic pressure; (h) chemotropism; (i) anthocyanins; and (j) environment. Not all of these theories are equally well substantiated but several of them are based upon quite extensive evidence brought out by the investigations of more than one competent worker.

*External structure.* — Formerly, perhaps, more stress was laid upon this factor of resistance than at present, although there are apparently well authenticated cases in which resistance to a particular disease is due to certain peculiarities of the external structure of the plant. The morphological characters most often mentioned in connection with disease resistance are those associated with the epidermal structure, such as number and size of the stomata, presence and thickness of the cuticle, presence of epidermal hairs, and the occurrence of waxes or "bloom" on the surface of the plant. Attention is called here to the fact that some of these items are mentioned again later under the topic "avoidance of disease."

In several instances the number and structure of stomata have been regarded as factors in disease resistance. Cobb (18) thought that the resistance of certain varieties of wheat to stem rust (*Puccinia graminis*) was due, partially at least, to the fact that the stomata of the resistant varieties were smaller than those of the susceptible varieties. Norton (59) found smaller stomata correlated with rust resistance in asparagus. Allen (2) noted that the stomatal opening is smaller in Kanred, a variety of wheat resistant to *Puccinia graminis tritici*, than in Baart, a very susceptible variety. According to Pool and McKay (63) the strong resistance of the immature leaves of beet to infection by *Cercospora beticola* Sacc. is due to the smallness of the stomata. On the other hand Eriksson and Henning (31) and Ward (83) found no evidence that resistance to *Puccinia dispersa* is related to stomatal structure.

Protective layers of cuticle or of cork have been credited with contributing to disease resistance in some cases. Immature tomato fruits are susceptible to rot caused by *Macrosporium tomato* while the older fruits are resistant, due, it is said (66), to the fact that the hyphae are unable to penetrate the thicker cuticle of the more mature fruits. Weimer and Harter (86), working with sweet potato rots, have demonstrated that the suberization of cell walls at the surface of wounds may prevent the entrance of decay organisms.

*Internal structure.* — Resistance in some cases appears to be related to the ease or difficulty with which the parasite can penetrate cell walls by enzymic digestion or otherwise. Since there are apparently chemical differences between the cell walls of different varieties of plants these differences are reflected in the degree of resistance exhibited by different varieties of host plants to a specific organism. A wall-dissolving or macerating enzyme has been reported as the factor which enables *Botrytis cinerea* to penetrate the tissues of susceptible plants (11, 12, 13). Jones (46) found that the soft-rot organism, *Bacillus carotovorus*, secretes an enzyme, pectinase, which enables it to break down the tissues of susceptible vegetables.

Another interesting case which throws some light on the nature of disease resistance is illustrated by the seedling-blight of corn and wheat caused by *Gibberella saubinetii*. While perhaps not belonging strictly in this category, this disease illustrates very nicely the interaction of environment, physiology and structure in determining the degree of resistance exhibited. Dickson (27, 28) has shown that resistance to this disease by both corn and wheat seedlings depends upon the readiness and rapidity with which the thin pectic cell walls of the young plants are built up and thickened with cellulose, lignin and suberin. This in turn depends upon the metabolism of the seedling and is concerned with the percentage and nature of the carbohydrates present. When the soluble carbohydrate wall-building substances are high the plant soon establishes a resistance. When pentosans are abundant and the wall-building carbohydrates deficient the fungus is favored and the plant shows less resistance. Thus resistance is correlated with the carbohydrate-pentosan ratio. Not only does this ratio vary in different strains of corn, for example, but in any particular strain the ratio varies with the temperature so that a strain of corn which is susceptible to seedling-blight at low temperatures may be very resistant at high temperatures due to the modification of this metabolic balance as a result of the increase in temperature.

Conant (19) has recently shown that a close correlation exists between the histological structure of tobacco roots and resistance to the root-rot fungus, *Thielavia basicola*. The root tips and elongating regions are relatively immune, apparently due to inability of the fungus to penetrate the epidermis. Farther back from the tip the epidermis is ruptured due to cambial activity, and since pericyclic activity lags behind cambial activity, no cork has yet appeared, consequently the roots are very susceptible in these regions. The fungus is unable to penetrate cork, therefore as soon as a phellogen appears and begins to lay down cork the fungus is barred from entrance. (See Chapter V for a discussion of the relation of temperature to plant diseases.)

Cell-sap acidity. — Some investigators have reported that in certain cases resistance seems to be due to acidity of the cell sap. One of the most recent cases recorded is that of the bacterial fruit spot of tomato caused by *Bacterium exitialium* G. and K. in which Gardner and Kendrick (35) found that seedlings, leaves, and green fruits were very susceptible to the disease while ripe fruits were resistant. This condition was found to be correlated with a higher acidity in the ripe fruit than in the other parts of the plant or in the green fruit. Tests made on culture media with different PH values substantiated this conclusion. Most of the evidence concerning the relation of cell-sap acidity to re-



sistance, however, is negative. Vavilov (79) investigated the resistance of varieties of wheat, oats and roses to rusts and mildew and found nothing to indicate that resistance was due to cell-sap acidity. Hurd (40, 41, 42) working on resistance in wheat to stem rust and to bunt found no evidence in either case that the H-ion concentration of the cell sap was correlated with disease resistance. Other workers have failed to find any correlation between cell-sap acidity and resistance to disease in case of the potato late blight (*Phytophthora infestans*) (47), leak of potato (*Pythium debaryanum*) (38), and potato wart (*Synchytrium endobioticum*) (87).

*Tannin.* — Cook and others (20, 21, 22, 23) have worked on the possible relation of tannin, tannic acid and other organic acids, to disease resistance. They found that tannin inhibited germination of spores. They were of the opinion that injury to the cell results in the production of tannin or a substance resembling tannin which has a toxic effect upon fungi.

*"Antagonism."* — This term is used for want of a better one to express a more or less definite antagonistic physiologic reaction between the host protoplasm and that of the parasite. In the highest type of obligate parasitism, such as that exhibited by the rusts, the most susceptible varieties of host plants are those in which the invaded cells are not killed but remain apparently healthy, for a time at least. On the other hand the resistant varieties are those in which the first cells to be invaded by the parasite are killed and apparently form a barrier to the further invasion of the fungus. Ward (84), working with a species of rust fungus, *Puccinia glumarum*, found that in case of susceptible varieties neither the hyphae nor the invaded cells show any evidence of injury until time for spore formation, while in resistant varieties the invading hyphae show signs of degeneration. From the evidence he concluded that the fungus was either being starved or poisoned. Several workers (55, 69, 70), investigating rust infection on various plants, agree that in all cases the fungus enters the substomatal chambers but that the further development of the fungal hyphae differs radically in susceptible and in resistant varieties. Allen (2) has contributed materially to a more exact knowledge of the factors of rust resistance in wheat. In a comparative study of the resistance of Baart, a susceptible variety, and Kanred, a resistant variety, to a strain of *Puccinia graminis tritici*, she found three factors of resistance in Kanred, namely, (a) the hyphae do not enter the stomata as readily as they do in Baart; (b) when the hyphae which do enter the substomatal chamber come in contact with the mesophyll cells, a reaction is set up which kills the host cell, and one or more toxic substances formed in the host cell diffuse out of the dead

cell and kill the haustoria and hyphae of the fungus in close proximity, whereas in Baart the haustoria penetrate the cells without apparent injury; (c) the diffusion of the toxic substances into the healthy host tissues immediately surrounding the invaded and killed cells is stopped by the thickening of the adjacent cell walls. Thus there are apparently three different types of factors to which rust resistance in Kanred wheat is due. Two of these have been previously discussed, namely, external structure, as exemplified by the stomatal structure in Kanred which does not permit the germ tubes to enter as readily or in as large numbers as in Baart; and internal structure, as illustrated by the thickened contact walls. The third factor consists of the antagonistic reaction wherein the presence of the fungus or certain enzymes or toxins secreted by it kill the invaded cells, and in which certain substances diffuse from the dead cell and kill the contiguous hyphae of the fungus which has just attacked it. In another paper Allen (3) describes the results of further investigations of rust resistance exhibited by Baart, Kanred and Min-dum wheats to certain strains of the stem rust fungus. In the main this work confirms her previous work with Baart and Kanred. In susceptible varieties the invaded cells apparently are stimulated to produce a greater food supply for the fungus, whereas in resistant varieties the host cells collapse and soon the haustoria in contact with these cells also die.

*Starvation.* — A few investigators have suspected that resistance might be due to failure of the fungus to secure the proper food for its further invasion of the host tissue. It was noted above that Ward (84) offered a starvation theory as a possible alternative explanation for rust resistance in wheat. Giddings (36) believed that food factors were primarily the cause of the development of resistance to rust (*Gymnosporangium juniperi-virginianae*) in a certain variety of apple. However, there does not at present seem to be enough convincing evidence to warrant listing this as a very important factor in disease resistance.

*Osmotic pressure.* — It has been suggested that a correlation exists between the osmotic pressure of the parasite and that of the host. In connection with his work on phanerogamic parasites MacDougal (51) concluded that the osmotic pressure of the parasite must be higher than that of the host. On the other hand, Hawkins (37), working with certain fungi causing decay in potatoes, apples and strawberries (*Fusarium*, *Botrytis*, *Sclerotinia*, *Rhizopus*, etc.), found that they would grow in culture media of much higher diffusion tension than that of the sap of their host plants.

*Chemotropism.* — The attempt to explain susceptibility to disease as due to the presence of a positively chemotropic substance in the cells

of susceptible plants has been refuted too often to bear much weight. It has been often demonstrated that fungi may penetrate many plants other than their recognized hosts. In such cases, however, the fungi soon cease to grow and the disease typical of their attack on susceptible hosts does not appear. Salmon (68) found that the haustoria of the wheat mildew (*Erysiphe graminis*) penetrated the epidermal cells of barley but soon died and no mildew symptoms appeared. According to Tisdale the cabbage yellows fungus, *Fusarium conglomerans*, may penetrate the root hairs of flax but never develops far enough to produce a wilt as is the case when the flax plant is attacked by *Fusarium lini*. Wiltshire (90) noted that the apple-scab fungus, *Venturia inaequalis*, could invade the pear and the pear-scab fungus was capable of entering the apple fruit but that neither could produce the disease on other than its own host. Gibson discovered that a large number of non-susceptible plants may be penetrated by the germ tubes of several rust fungi but that no further development occurs. These instances indicate that resistance is not a question of inability to enter the host but of inability to develop and cause typical symptoms after penetration has been effected.

*Anthocyanins*. — Many instances of the supposed resistant effect of certain anthocyan pigments have been noted in the literature of disease resistance. Red potatoes have been credited (45) with being more resistant than white-skinned varieties. Red apples have been considered more resistant to scab than others. The experimentally proved cases of resistance due to anthocyan pigments are few. Fromme and Wingard (33) found that varieties of beans with solid red or red mottled seed were resistant to bean rust (*Uromyces appendiculatus*) whereas the white-seeded varieties were susceptible. Walker (80, 81) offers convincing evidence that red and yellow skinned onions are very resistant to onion smudge (*colletotrichum circinans*) while the white skinned varieties are susceptible.

*Environment*. — It is difficult to draw a sharp line between the effect of environment as influencing real resistance and the part it plays in the avoidance of disease. It undoubtedly wields an influence in both cases. Where the environment brings about a change in either the external or internal structure of the plant or in its physiological reactions sufficient to make the plant either more or less resistant, then it may truly be said to influence the resistance and susceptibility of a plant. Tisdale (76) has shown the influence of high temperatures upon the susceptibility of flax to wilt (*Fusarium lini*). Dickson has pointed out the effect of high temperature in rendering corn more resistant to seedling-blight, as was noted above in another connection.

**The avoidance of disease.** — Sometimes it is difficult to distinguish clearly between truly resistant plants and those which might be called "disease-escaping." Perhaps the simplest cases of escaping or avoiding diseases in plants are those which may be attributed to changes in the environmental factors. Weather conditions have great influence in decreasing or increasing the amount of infection. During a wet spring and early summer a susceptible variety of apple may develop a high percentage of scab, whereas during a dry spring the same variety may develop very little scab. This does not mean that this apple is truly resistant to scab but that, due to conditions in this particular year, it "escaped" scab infection to a great extent. Season has equal influence in this respect. A variety of wheat, which under favorable conditions is quite susceptible to smut, may, in some sections of the country, "escape" smut infection if sown either earlier or later than the usual seeding time. Orton (61) calls attention to the fact that Early Ohio potatoes avoid late-blight by reason of their early maturing habits, and an early cowpea escapes the *Fusarium* wilt for the same reason. He also mentions cases of disease endurance in certain varieties of plants due to various characters such as vigor, hardy structure and drouth-resistant qualities.

Other more extreme cases may be cited to illustrate the difficulty of determining just where disease avoidance ceases and true resistance begins. Appel (5) suggests that some varieties of potatoes seem to be more resistant to late-blight (*Phytophthora infestans*) than others, whereas they only escape the disease more readily than other varieties because their leaves dry more quickly after a rain, thus offering less favorable conditions for the germination of the spores of the late-blight fungus. That is, quick-drying varieties are more likely to escape infection than slow-drying ones and thus appear to be less susceptible. Since the characters which determine the rate of drying are evidently morphological, the question arises as to why this case should not be classed under the type of resistance due to external structure as discussed in a previous paragraph.

Another instance of the inter-relation of various factors such as environment and structure in rendering a plant able to withstand disease is illustrated by the work of Freeman (32). He grew barley in soils of different degrees of alkalinity and inoculated these plants with rust. The barley plants grown in the more alkaline soil showed less rust than those grown in the less alkaline soil. The few rust pustules that did appear on the latter plants, however, were large and vigorous looking sori. He explained this difference as being due to the greater development of "bloom" on the barley foliage when grown in the soil of

stronger alkalinity. This "bloom" or wax on the surface of the leaves caused the water to roll off the foliage more readily, hence there were fewer rust infections on these plants than on those grown under conditions that produced less "bloom." Thus it can be seen that this morphological character, namely, excessive "bloom" or wax on the surface of the host, enabled it to escape the rust more often than the plants with less wax, but that these "escaping" plants were not really immune to the disease as shown by the vigorous development of the rust wherever infections did occur. Some would perhaps extend the use of the term "disease-escaping" to include plants which remain free from disease for any reason other than on account of the peculiar physiological or chemical make-up of the living cell. This would reduce all true resistance or immunity, then, to a question of chemical bodies in the host plant. This view seems to be extreme, however, and the more reasonable view is, perhaps, to include distinct structural characters, both internal and external, among the factors to which true resistance may be due, in addition to those physiological or chemical factors which certainly are to be reckoned with in determining the basis of resistance.

**Means of securing resistant varieties.** — There are several ways of securing varieties of plants which are disease-resistant. The methods most often used are: (a) by introductions; (b) by selections; and (c) by hybridization.

*Introductions.* — It sometimes happens that new varieties or strains of some of our crops, when brought in from foreign countries, prove to be more resistant to certain diseases than varieties which have been grown here for a long time. If these introduced resistant plants are not desirable from other standpoints they may often be used with profit as a starting point for selecting or breeding desirable strains of disease-resistant crops.

*Selections.* — The principle of selection may be applied either to the use of a particularly resistant variety or strain in preference to a more susceptible variety, or to the selection of resistant individuals from within a variety. The latter method was well illustrated in Wisconsin where strains of wilt-resistant cabbage were developed by saving, as seed plants, individual cabbages which withstood the disease in fields where most of the plants succumbed because of the severe infestation of the soil with the wilt organism.

*Hybridization.* — Sometimes the process of selection alone does not result in the type of plants desired. Perhaps when the investigator finds a variety or an individual which possesses the desired resistance to disease he discovers that other desirable qualities are lacking. He may

find a variety of wheat immune to smut but this wheat may be good for nothing after all. It may be a poor yielder, have very poor milling qualities and possess many other undesirable characters. But if it is truly immune to smut he may be able to cross it with a variety which has all the other desirable qualities and thus finally breed up a strain which is satisfactory in all respects including immunity to smut.

**Genetics of immunity.** — In order to go far in the process of securing immune varieties by hybridization one should have some training in genetics and plant breeding. Resistance and immunity are subject to the laws of heredity. For example, Biffen (8) found that in case of certain varieties of wheat which are resistant to the stripe rust fungus, *Puccinia glumarum*, rust resistance is a unit character which, in crosses between resistant and susceptible varieties, behaves as a recessive Mendelian character. Armstrong (6) also worked with stripe rust and found that resistance and susceptibility behave as unit characters and are inherited in accordance with the simple Mendelian law; that in the  $F_2$  generation susceptible and immune individuals are segregated in the ratio of three to one; that the immune  $F_2$  plants breed true to that character; and that among the susceptible plants of the  $F_2$  generation one-third are homozygous for susceptibility and breed true, while two-thirds of them are heterozygous for susceptibility giving rise to progeny in which susceptible and resistant plants occur in the proportion of 3 : 1 as in the  $F_2$  generation. Melchers and Parker (57) made crosses using three varieties of winter wheat which were resistant to a strain of stem rust (*Puccinia graminis tritici*), and three susceptible varieties of spring wheat. On testing succeeding generations resulting from these crosses they found that, with these varieties, resistance is dominant and susceptibility is recessive.

It will be noted in the cases cited above that rust-resistance is a recessive character in one instance and a dominant character in the other. This seems to indicate that there is no uniform rule governing the inheritance of disease-resistance in plants. There is a vast accumulation of literature dealing with the problems of genetics in general and a goodly percentage of these publications deal with the particular problems of breeding plants for resistance to plant disease. The list of titles among the references at the end of this chapter will serve to indicate some of the various problems connected with investigations of this kind.

**Difficulties met in developing resistant varieties.** — The investigator who attempts to develop varieties of any particular crop resistant or immune to any specific disease is likely to encounter some difficult problems before he can hope for complete success. There are at least

three of these serious obstacles, any one or all of which must be met and overcome before satisfactory varieties can be developed by selection and hybridization. These are the facts: (a) that immunity to disease is by no means necessarily associated with other desirable qualities in a particular variety; (b) that a plant may be found which is resistant to one disease but very susceptible to another disease of equal or greater danger; and (c) that there are, in many cases, several biologic strains of the same organism and while a variety of the host may be resistant to certain strains, it may be very susceptible to another strain or strains.

*Immunity must coincide with other desirable characters.* — Reimer (65) found certain varieties of Chinese pears which were practically immune to fire-blight but these pears were of no commercial value. His problem was to use these varieties of pears as a basis for building up a pear that would be immune to blight and at the same time have a high commercial value. It is manifest that this is likely to be a much more difficult task with the pear than a similar problem with some annual or biennial plant such as bean, corn, cabbage, etc., would be. As is well known pears are ordinarily propagated vegetatively by budding or grafting and the only way to secure any immediate results from the immune varieties is to use them for grafting or budding stock on which to grow the commercial varieties. The alternative is to hybridize and trust to luck that after many years' waiting a seedling may fortunately be found which will combine the characters of blight resistance and commercial desirability.

*Resistance to two or more diseases not always coincident.* — The value of finding or developing a resistant variety or strain of any particular crop would be greatly enhanced if it were true that a variety which is resistant to one disease were likewise resistant to all other diseases to which the species is subject. While exceptionally hardy varieties which are more or less resistant to diseases in general are sometimes developed, such as a variety of cowpea resistant to both wilt and root-rot, it is equally true that such coincidence is not always encountered. A variety of apple which is resistant to scab may not necessarily be resistant to all other apple diseases. A variety of wheat resistant to smut may be susceptible to rust, scab or root-rot; although some wheat strains which seem to be resistant to most wheat diseases are known. When we take these facts into consideration, along with the fact that our crop plants are nearly always subject to more than one disease — some of them, such as the potato, to very many — it becomes evident that the problem is greatly complicated. It can readily be seen that even if a variety is developed which exhibits resistance to one disease, it is by

no means certain that it will not be susceptible to other diseases of equal or greater importance.

*Biologic races.* — Evidence has been accumulating for many years that some species of plant pathogens are composed of more than one strain or variety. Perhaps the most classical example of this phenomenon that we have today is that of the fungus causing black stem rust of wheat. Not only has it been shown that this species, *Puccinia graminis*, has a separate strain for wheat, oats, and rye, but it has also been shown that the wheat strain is again subdivided into many different strains or forms. Hayes, Stakman and Aamodt (39) stated in 1925 that there were then known forty or more different strains or physiologic forms of the stem rust fungus. Leach (50) found at least eight distinct biologic forms of the bean anthracnose fungus, *Colletotrichum lindemuthianum*.

It can readily be seen that the existence of so many different forms or varieties of a species of parasite complicates the situation very greatly when an attempt is made to select or breed a resistant variety of the host plant. If a variety should be selected or developed which proves resistant to a certain strain of the parasite, there is no assurance whatever that this variety will also be resistant to all the other strains or biologic forms of the organism. However, in spite of all the difficulties enumerated, it still remains true that the development of resistance and immunity in plants offers a wide field for research and constitutes the only avenue of approach for the control of certain plant diseases.

**"Artificial" immunization.** — The question is frequently raised as to whether plants can be immunized to disease in the same manner that animals and man are rendered immune to certain diseases by the use of serums or the production of anti-bodies. No progress has yet been made in this direction and it is doubtful if this ever can be done with plants as it is done in the animal kingdom. The structure of plants is so different from that of animals, especially as regards a circulatory system, that it does not seem likely that much success can ever be attained in that direction. In so far as can be predicted at the present time, it seems probable that the attainment of immunity in plants will have to come through selection and hybridization as discussed above rather than through the administration of serums or any means now used in human medicine.

#### REVIEW QUESTIONS

1. Why is it desirable to have disease-resistant varieties of plants?
2. What is the difference between disease avoidance and disease resistance?
3. Mention two general headings under which all factors responsible for true resistance in plants may be listed. Under each heading mention the specific factors that have been suggested in different cases by various workers.



4. What factors may be responsible for a plant's escaping a disease to which it is not truly resistant?
5. Discuss the means of securing resistant varieties.
6. What difficulties must be overcome in developing resistant varieties?

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## CHAPTER XII

### RELATION OF INSECTS TO PLANT DISEASES

The insects as a group cause an immense amount of damage to plant life. Such insects as grasshoppers, borers, codling moths, caterpillars, aphids, weevils, etc., annually take an enormous toll in destroyed or damaged crops. In defining the scope of plant pathology some writers have included insects and their depredations as a legitimate part of the general field of plant pathological investigations. This is especially true of European workers, some of whom, at least, contend that there is no fundamental difference between ordinary insect attacks and the work of fungi and bacteria. All endanger the life of the plant or reduce its economic value. In the broadest sense this, of course, is true. In the United States, however, as the various agricultural sciences have developed, in both the Federal Department of Agriculture and in the state institutions and experiment stations, a rather strict segregation of subject matter in separate bureaus and departments has occurred. Thus in this country we have on the one hand the economic entomologists whose field includes the insects and the plant injuries caused by them, and on the other hand the plant pathologists who confine their attention largely to non-insect troubles such as the fungous, bacterial and virus diseases together with certain non-parasitic troubles which are included by common consent in the subject of plant pathology. This division of labor, while it may not be strictly logical from certain points of view, allows for greater specialization and is undoubtedly advantageous in some respects. Under such an arrangement the plant pathologist is freed from giving attention to the great army of insects which the entomologist has undertaken to control but he cannot avoid entirely contact with the insect world.

A very important phase of investigational work in plant pathology has to do with the dissemination of plant diseases by insects. Particularly among the bacterial and the virus diseases of plants, insects play an important rôle in the transmission of the causal agent. Some fungi also may be carried by insects. Thus the plant pathologist must meet and solve, or at least help to solve, certain problems connected with insects in their relation to the dissemination of infectious plant diseases. The following outline indicates the phases of this subject which are of most interest here (41).

- I. Insect wounds through which diseases are introduced without transmission by the wounding agent.
- II. Transmission of disease-producing agents by insects.
  - A. Types of Transmission.
    1. External transmission.
      - a. Direct inoculation.
      - b. Accidental inoculation.
    2. Internal transmission.
      - a. Mechanical.
      - b. Biological.
  - B. Number of diseases disseminated by insects.
  - C. Systematic relation of insect carriers.
  - D. Problems connected with insect transmission.

**Causal agents entering through insect wounds but not carried by insects.** — It is a well-known fact that the causal agents, both fungous and bacterial, of many of the parasitic plant diseases enter largely or entirely through wounds of various sorts in the host plant. Wounds in plants are due to various causes, as, animals, insects, fire, meteorological factors and man. In all of these types of wounds, causal organisms may find entrance, even though they might not have been carried there by the agent causing the wound. Referring to wound invasion in general, the wood-rotting fungi, especially those causing heart-rots, usually gain entrance through rather large wounds caused by one or another of the agencies mentioned above, but are not as a rule carried by these agencies.

With particular reference to insect-made wounds there are a large number of examples which may be cited. It is a matter of common occurrence that rots, such as soft rot caused by *Penicillium* sp., gain entrance to apple fruits through the burrows of the codling moth. The fungus, *Colletotrichum falcatum* Went., which causes the red rot of sugar cane, probably invades the canes through wounds made by the Hawaiian sugar cane borer (28). Whetzel (66) says that onion plants which have been weakened by the attacks of thrips and of maggots are more susceptible to infection by the blight fungus, *Peronospora schleideni* Ung., than plants free from these insects. The chestnut blight fungus, *Endothia parasitica* (34, 43), and the pecan scab organism, *Fusicladium effusum* (65), are said to infect through insect wounds at times. It is not claimed that these organisms are never carried by insects. Probably some of them are insect disseminated. The point is that they may gain entrance through insect wounds without having been carried there by the wounding agent.

**Insect transmission of disease-producing agents.** — Many instances of insect transmission of pathogenic organisms are already known and future research will undoubtedly bring to light many other cases. There are two general types of insect transmission, namely, (A) that in which the organism is carried *externally* on some part of the insect, as feet or mouth parts, and (B) the type in which the parasite is carried *internally*. In case of external transmission the causal agent may be (a) directly inoculated into the host by the disseminating agent, or (b) the inoculation may take place accidentally. There are also two types of internal transmission, (a) mechanical, and (b) biological. In mechanical internal transmission the causal agent is merely passed through the digestive tract without undergoing multiplication or other important change. In biological internal transmission the causal agent may pass a considerable part of its life history within the insect, undergoing multiplication, hibernation or other important changes.

**External transmission.** — Examples of this type of transmission occur among both animal and plant diseases, but we shall consider only plant diseases in this discussion. Both fungi and bacteria are externally disseminated by insects and either directly or accidentally inoculated into plants. Probably in some cases both direct and accidental inoculation occur in the same disease.

(a) *Direct inoculation.* — Several bacterial diseases of plants are thought to be carried externally on insects and directly inoculated into plants. The bacterial wilt of potato and other solanaceous plants caused by *Bacterium solanacearum* has been experimentally transmitted (51) by using the Colorado potato beetle as a transmitting agent. The cabbage black-rot organism, *Bacterium campestre* (52, 53, 54) and a bacterium causing a rot of lettuce (7) are said to be transmitted by insects in this manner. Aphids and leaf-hoppers (67) and a bark-borer (24, 25) are thought to carry the fire-blight organism, *Bacillus amylovorus* and introduce it into the host trees.

Fungous pathogenes also may be externally carried from diseased to healthy plants. Scott and Ayers (46) believe that sucking insects of the squash-bug family not only carry spores of the brown-rot fungus but that they may actually insert them into the peach. Scott and Quaintance (47) hold the plum curculio responsible for the spread of the brown-rot fungus in some cases. Heald (18) felt quite certain that the fungus, *Sporotrichum anthophilum* Peck, which causes a bud-rot of carnations, was carried and inoculated into the buds by a species of mite. Grossenbacher and Duggar (17) suggest that the currant borer may have some relation to the dissemination of the currant cane-blight fungus, *Botryosphaeria ribis*, and its inoculation into currant plants.



(b) *Accidental inoculation.* — Perhaps the fire-blight organism is one of the best-known examples of an insect-transmitted pathogene. In addition to the sucking or biting insects mentioned above which may transmit the fire-blight bacillus, there are other insects such as bees, wasps and flies which undoubtedly are responsible for a great deal of accidental inoculation in connection with this disease. These insects transmit the organism from exuding cankers to wounds or blossoms, or from blossom to blossom. Infection may take place in the nectaries and probably in the leaves (19) in the absence of wounds, from bacteria accidentally deposited in these places by insects. Waite (62) first demonstrated that the fire-blight organism is transported from flower to flower by bees and other insects and that blossom blight is caused only in this way. More recently Gossard and Walton (14, 15) have contributed extensive evidence as to the work of honey bees in disseminating the fire-blight bacillus and the ability of the organism to survive in honey for a certain length of time.

There are several examples of fungous diseases of plants in which external insect dissemination and accidental infection occur. Some investigators have thought that certain rusts (13, 23) and smuts (6, 22) are transmitted to some extent by insects. Clinton (9) held certain small flies responsible for considerable dispersal of the apple bitter-rot fungus. Some investigators have thought that insects are largely responsible for the dissemination of the chestnut blight fungus, *Endothia parasitica* (Murr.) P. J. and H. W. And. (20, 32, 33, 34). Others (10) have doubted the importance of insects as carriers of this parasite. The beet leaf-spot fungus, *Cercospora beticola* Sacc. seems to be carried by certain insects (30).

**Internal transmission.** — Instances of this type of insect transmission are known among the bacterial, the fungous, and the virus diseases. In some cases it is quite evident that the organisms are carried internally in a purely mechanical manner without any indication of a biological relationship. In other cases there is every evidence of the existence of a close biological relationship between the disease-producing organism and its insect carrier. In still other cases disease organisms are known to be carried by insects, but definite proof of their exact status, that is, whether the transmission is mechanical or biologic, is lacking.

(a) *Mechanical transmission.* — The pores of several species of parasitic fungi are capable of passing through the intestinal tract of insects in a viable condition. That certain flies carry ergot spores not only on their bodies but in the alimentary tract and deposit them on grasses was demonstrated by Mercier (31). Parrott and Fulton (36)

claim that the snowy tree cricket carries internally the spores of the raspberry cane-blight fungus, *Leptosphaeria coniothyrium* (Fekl.) Sacc. Gravatt and Posey (16) showed that the gypsy moth larvae carry large numbers of spores of the white-pine blister-rust fungus, *Cronartium ribicola* Fischer, on and within their bodies. It should be borne in mind that the mere discovery of viable fungous spores in the alimentary tract or feces of insects does not prove that the insect transmits the disease in this manner but it at least points to the possibility or even probability of such transmission. In a few cases enough evidence has been collected to establish beyond a reasonable doubt the occurrence of internal mechanical transmission.

(b) *Biological transmission.*— There are a few bacterial diseases of plants in which the causal organism apparently has a symbiotic relationship with some insect. From the work of several investigators (38, 39, 40, 49, 50) there is strong evidence of an internal biological relationship existing between the organism causing bacterial wilt of cucurbits, *Bacillus tracheiphilus* E. F. S., and the striped and spotted cucumber beetles. Petri (37) affirms that the olive-knot organism, *Bacterium savastanoi* E. F. S., passes a part of its life cycle in the olive fly, *Dacus oleae* Rossi.

Among the fungi some yeasts and molds are said to live and even multiply within certain insects (5). Schneider-Orelli (44) found an interesting relationship existing between the beetle, *Xyleborus pyri* Peck, and the fungus *Monilia candida* Hartig. He found the living fungus always present in the crop of the female beetles. When the insect bores into a tree it apparently "plants" this fungus for shortly afterward the tunnel becomes lined with a white mycelial growth of the fungus. The larvae of the beetle apparently eat the fungus.

One of the most important classes of plant diseases depending upon insects for dissemination is the group generally known as the virus diseases. Many of these diseases are transmitted by insects, especially aphids. The curly-top disease of sugar beets is probably the most outstanding case among the virus troubles. This malady is known to be transmitted by a single specific insect, the leaf-hopper, *Eutettix tenella* Baker (4, 48), and so far as has been demonstrated up to the present time cannot be transmitted in any other way. In this case the relationship existing between insect and contagium is probably biological. There is some indication that the curly-top virus requires a brief incubation period of at least a few hours in the insect before it can be transmitted. Aster yellows is another disease resembling curly-top in that only a single species of insect can transmit the yellows contagium. In this case the incubation period is longer, ranging from six days to

two weeks (27). Many other instances of insect transmission of viruses have been demonstrated but the exact status, whether biological or merely mechanical, has not been definitely determined. Allard (1, 2, 3) showed that tobacco mosaic is carried by aphids. Taubenhaus (61) reported that mosaic of sweet pea is transmitted by aphids. Doolittle (11) and Jagger (21) demonstrated that aphids carry cucumber mosaic, and Doolittle and Gilbert (12) showed that the striped cucumber beetles also carry this virus. Schultz and others (45) succeeded in proving that the virus of potato mosaic may be disseminated by aphids. From the fact that many of these virus troubles can be artificially transmitted by man in various ways, it would seem logical to conclude that the insect transmission of such diseases is probably mechanical rather than biological.

The above discussion by no means includes all known examples of insect transmission of plant diseases. However, it is sufficient to show the importance of this phase of plant pathology. Biological internal transmission is one of the most interesting phases of this subject but one which is at the same time difficult to investigate. In the field of human and animal medicine great strides have been made in the acquiring of knowledge of certain diseases which are transmitted in this way, and plant pathologists have profited much by the experience of the animal pathologists. However, much remains to be learned about the various relations of insects to plant diseases. Probably the most vital phase of the whole subject at present is the part which insects play in the dissemination of the virus diseases.

**Number of diseases transmitted by insects.** — In 1922, Rand (42) reported that the total number of plant diseases definitely known to be insect transmitted amounted to eighty-five, with forty-one other strongly suspected or doubtful cases. Of the definitely established cases, nine are of bacterial origin, sixteen are due to fungi, one is caused by a protozoan, and fifty-nine are virus diseases. In the latter case, however, the number of hosts found infected with virus troubles is referred to rather than the number of distinct virus diseases, since it is now known that a single virus may attack more than one species of plant, and also that a single host species may be attacked by more than one virus. Thus the number of different species of plants affected with virus troubles is no indication of the number of different virus diseases in existence. The number of known hosts and also of distinct virus diseases is constantly increasing.

**Systematic relations of insect carriers.** — There are apparently about three different conditions as regards the systematic relationships of insect carriers. In such cases as the curly-top of sugar beets and other

plants only a single species of insect is able to carry the contagium. In contrast with this narrow limitation of carrying ability, there are those diseases such as fire-blight in which the insect contact may be considered entirely accidental. Here any insect which chances to come in contact with the bacterial ooze may carry the germs to another host. There is no special adaptation of any kind and the causal agent has no symbiotic relationship whatever to the insect carrier. An intermediate condition is found where transmission may be confined to a certain species of insect, not because of any biological relationship nor because no other insect could carry the contagium but simply because of the fact that this particular insect is associated with the host in such manner as easily to carry the causal agent or inoculate it into the host. The fact that certain of the virus diseases seem to be carried in nature only by aphids appears to be an illustration of the latter condition, since in some of these cases the disease can be transmitted artificially, indicating that a strict biological relationship with the aphid carriers does not exist.

**Problems connected with insect transmission.**—Rand and Pierce (41) have given a brief résumé of the problems connected with insect transmission of plant diseases and later Gardner (42) has given a very comprehensive statement of these problems with especial emphasis on insects as related to the so-called virus diseases. Problems of this sort naturally require a certain knowledge of insects as well as of plant diseases proper. This calls for the coöperation of entomologists in certain phases of the problem. Thus it seems that some phases of the problem can be solved by plant pathologists working alone, other phases by entomologists working alone and still a third type of problem may call for the combined effort of plant pathologist and entomologist working together.

The particular sphere of the plant pathologist has to do with such questions as the nature of the causal agent; the transmitting agent, insect or otherwise; the host range of the parasite or virus; and the conditions under which infection occurs. The entomologists should be able to contribute valuable information on effective control of the carrier species of insects; more detailed life history studies on incriminated species; histological and physiological studies of viruliferous individuals in the insect population; and investigation of the possibility that the insect carriers may be partial to certain strains or varieties of the host plant.

*Specific questions.*—Many specific questions concerning insect-transmitted diseases still remain unanswered. Some of the questions in the following list will apply to any type of disease, fungous, bacterial or virus, while others pertain more particularly to the virus diseases. The

list is by no means complete but is suggestive of the type of problems yet remaining to be solved in this field.

1. In any particular insect-transmitted disease is there an obligate symbiotic relationship of some sort existing between the pathogene and the insect carrier or is the transmission merely accidental?

2. Is biological insect transmission limited to those cases in which artificial inoculations are impossible?

3. In cases where the relationship between insect and causal agency is apparently not biological, as proved by successful artificial inoculation, why is natural transmission apparently limited to certain species of insects? For example, why are aphids the only known insect-carriers of certain mosaic troubles which have been artificially transmitted?

4. Is it necessary for a carrier insect to puncture certain tissues in order to transmit the infective principle?

5. How long must an insect feed upon a diseased plant before it acquires the ability to transmit the contagium to a healthy plant?

6. After feeding on a diseased plant how long does the insect retain the infective capacity?

7. What is the minimum length of time a carrier insect must feed upon a healthy plant in order to cause infection?

8. Does the insect carry the contagium through hibernation?

(For a detailed discussion of problems in connection with insect transmission of the virus diseases in particular, see reference 42 following.)

#### REVIEW QUESTIONS

1. Name the two general types of insect dissemination of plant diseases.
2. Distinguish between *mechanical* and *biological* internal transmission.
3. Give examples of diseases in which a biological relationship exists between the causal agent and the insect which transmits it.
4. What different types of causal agents may be insect-transmitted?
5. Mention some of the important unsolved problems connected with insect transmission of plant diseases.

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## CHAPTER XIII

### STORAGE, TRANSPORTATION AND MARKET PROBLEMS

It has been customary to think of plant diseases as largely if not entirely confined to growing crops in the field. But by no means all crops are safe from disease when harvested. Indeed, with a great many plant products, and especially in reference to certain types of diseases, the menace after harvest is as great as, and in many cases greater than, it was while the crop was still growing in the field. Enormous losses are sometimes sustained in certain kinds of produce, especially fruits and vegetables, after they are placed in storage or while in transit, or even after arrival at the market. Most of the losses occurring after harvest are due to organisms of decay but some of the non-parasitic or "physiological" diseases develop in storage or transportation.

A few examples of the enormous losses suffered in transit will serve to show the seriousness and importance of this phase of plant pathology. Shear (19) quotes the American Association of Refrigeration as authority for the statement that in 1914 the amount of claims paid by 180 railroads for loss of perishable freight was \$4,977,383.09. Over one-half of this amount was for vegetables and fruits. Link and Gardner (10) cite instances of losses occurring in transit. During the season of 1918 a loss of 2500 carloads of watermelons valued at \$1,250,000 was sustained in the shipments from four states. In one week 2500 hampers of head lettuce from Louisiana were rejected at the car in Chicago. Losses of 15 to 25 per cent commonly occur in shipments of Cuban pineapples on account of Thielaviopsis rot.

Some of the factors which must be considered in a study of the disease problems related to storage, transportation and marketing are suggested in the following outline:

- I. Types of injury occurring.
  - A. Parasitic diseases.
    1. Blemishes.
    2. Rots.
  - B. Non-parasitic troubles.
    1. Mechanical injuries.
      - a. Relation to parasitic diseases.
    2. "Physiological" diseases.

- II. Time and place of origin.
  - A. In the field before harvest.
  - B. In transit, market or storage.
- III. Contributing causes.
  - A. Lack of field control.
  - B. Careless handling.
  - C. Lack of sanitation.
  - D. Environmental factors.
    - 1. Soil and climatic conditions.
    - 2. Temperature, humidity and ventilation.
  - E. Condition at harvesting.
- IV. Control.
  - A. Determination of the cause.
  - B. Education of the grower, packer and shipper.
  - C. Inspection service.

**Types of injury.** — Many different types of injury are encountered under storage, transportation or market conditions. These troubles may be classified on various bases. From the standpoint of cause they are of either parasitic or non-parasitic origin. On basis of symptoms they range all the way from the most inconspicuous blemishes of little economic importance to the rapid-spreading and very destructive rots. The symptoms may be entirely internal with no visible external indications of trouble, or they may be confined more or less to the surface of the affected parts.

*Parasitic diseases.* — There are a considerable number of parasitic maladies which result in **blemishes** of various sorts without actually destroying the fruits or vegetables affected. While these blemishes in many cases do not seriously affect the food value of the products on which they occur, yet they reduce the market value and thus cause material loss in a great many instances. Well-known examples of such diseases are apple-scab, potato-scab, peach-scab, apple sooty-blotch, bacterial bean blight, coryneum peach blight, onion smudge or anthracnose, and the *Rhizoctonia sclerotia* on potato tubers. In addition to the lower grades assigned to such products with the consequent depreciation in market value, there is another item of loss which sometimes follows injuries of this milder type. It is not uncommon for secondary rot-producing organisms to gain entrance at some of the blemished spots and cause still further loss. Sometimes these secondary rots may cause injury far in excess of the original trouble.

Of all the parasitic plant diseases which enter into the problems of storage, transportation and marketing, those of the rot type are far

more destructive than those of the blemish type. Some of these rot diseases are distinctly field diseases which may also show up in storage, in transit or on the market, while others are more strictly confined to the latter and are not usually encountered on the growing crop in the field. ✓ Among the former may be mentioned brown-rot of stone fruits, bitter-rot, black-rot and northwestern anthracnose of apples, black-rot of grapes, bean anthracnose, buckeye rot of tomato, and watermelon anthracnose. Some of the diseases which are encountered more often after the crop is harvested are blue-mold rot (*Penicillium*), black-mold rot (*Rhizopus*), and gray-mold rot (*Botrytis*). Such rots as the slimy soft-rots caused by bacteria of the *Bacillus carotovorus* type and by *Sclerotinia sclerotiorum* are serious on many vegetables in storage or in transit.

*Non-parasitic troubles.* — Among the non-parasitic troubles one type which assumes great importance in this connection is that of **mechanical injuries**. ✓ Most injuries of this sort, unless extensive, are not very serious in themselves, but their rôle in affording easy access to certain rot organisms makes them of great importance in the control problem. Some of the typical storage or market rots would not attack at all in the absence of wounds while others would be less serious if all produce were free from mechanical injuries of every kind. ✓

Other non-parasitic disorders which show up in storage or market are the so-called "**physiological**" diseases. ✓ Some of these disorders show up conspicuously in storage or market even though they may have their origin in the field before harvest. ✓ Examples of such diseases are bitter-pit of apples, internal brown-spot and hollow-heart of potatoes, and point-rot of tomatoes. Other troubles of this sort may originate entirely in storage or transit, as apple-scald and black-heart of potato.

**Time and place of origin.** — As to place of origin most of the storage and transportation diseases fall into one of two classes: (a) those originating in the field before harvest, and (b) those that originate after harvest. There are probably a few cases in which the trouble may have its origin either before or after harvest. ✓

*In the field before harvest.* — All of the diseases listed above as causing blemishes which increase not at all or only very slowly in storage have their origin in the field. In addition to these many of the distinct rots are also of field origin, that is, they are diseases which naturally occur on the crop during the growing season but carry over in one way or another after harvesting and thus become of importance also on the stored or marketed product. ✓ There are two typical conditions in which the disease may exist at harvest time. Incipient infection may already have occurred but may not have progressed far enough to be visible at

all, or if visible, the lesions may escape notice or be considered so small as to be of no consequence. On the other hand infection may not actually have taken place but the spores or other propagative parts of the organism may be present on the fruit or vegetables and be carried thereon into storage or transit where infection may later take place if conditions are favorable. In either case the disease can be said to originate in the field since that was the source of the inoculum even if actual infection did not take place until long after harvest. ✍

*In transit, market or storage.* — In the broadest sense of the term this class might be considered to include the second group mentioned in the paragraph above. If we eliminate all diseases which occur regularly in the field as diseases of production which may later show up also in storage, etc., we thus limit the class under consideration to such molds and rots as may appear after harvest without regard to their existence on the particular crop in the field before harvest. Simply as a matter of classification alone, this may not be an important point but when we come to consider control measures the exact source of the disease may make a great deal of difference. This point will be discussed later.

**Contributing causes of disease in storage, etc.** — In view of what has been said above it can readily be seen that there are several factors concerned in causing the various transportation and storage diseases of perishable plant products.

*Lack of field control.* — It was shown above that many diseases found on the market, etc., are there only because the same diseases were present in the field before harvest. Obviously, then, if these diseases had been properly controlled by the grower, no trouble would have been experienced from this source later.

*Careless handling.* — Since some of the diseases under discussion can gain a foothold only through wounds, and since infection and spread in many others are favored by injury of any sort, it follows that anything tending to cause mechanical injury to the tissues will be a contributing cause of disease. ✍ While with our present facilities for handling plant products it would probably be too much to expect the elimination of all mechanical injury, yet with proper care in harvesting, packing, stowing and transporting it should be possible to reduce such injuries to a very small minimum and thus prevent a large per cent of disease formerly due to this cause.

*Lack of sanitation.* — That type of disease in which the causal organism is ever present or can exist for some time in storage quarters or in carriers will be hard to stamp out unless proper sanitary measures are taken to eliminate any spores or bacteria which may remain in the cars or warehouses, etc. For example, if a carload of fruit or vegetables

suffers severely from *Penicillium* or *Rhizopus* rot and this same car is used again soon for shipping another load of a susceptible crop without first thoroughly disinfecting the car, there is great danger that the next shipment will suffer likewise.

*Environmental factors.* — Such factors as temperature, humidity and ventilation have their effect on both the parasitic and the “physiological” diseases. The parasitic diseases in general develop more slowly or not at all as the temperature and humidity approach the zero point. However, there is a difference in the minimum and optimum temperatures for various fungi and some of them will keep on growing at temperatures ordinarily obtaining in cold storage or in refrigerator cars. In general, however, if the temperature is allowed to rise above a safe minimum and excessive humidity exists, loss is certain to occur from parasitic diseases. The non-parasitic (physiological) diseases which develop in storage or shipment are mostly due either to high temperatures or poor ventilation or both. Examples are apple-scald and black-heart of potato. Freezing of course is an exception to this rule. The physiological diseases which originate in the field are usually due to adverse soil, weather or climatic conditions. This type is illustrated by the internal brown-spot and hollow-heart of potatoes and the bitter-pit of apples.

*Condition at harvesting.* — This not only includes the presence or absence of incipient infection covered under the topic “Lack of field control” but also such conditions as over-ripeness, lack of maturity, and any other variation from prime condition due to unfavorable growing conditions of any kind.

*Control.* — The elimination of loss from disease in storage, transit and market involves three chief factors, namely, determination of the cause of the trouble; education of everyone concerned including the grower, packer, shipper and transportation companies as to the means of control; and the proper training of inspectors whose duty it is to aid in the enforcement of control measures.

*Cause.* — It is necessary to determine the cause of a particular trouble and especially its place of origin in order to be able to place responsibility for its control. For example, if it is a case of brown-rot of peaches, that places the blame primarily upon the grower because this disease is one of field origin and if field control is adequate there will be little danger of an outbreak in transit. Of course good refrigeration will help to keep down the spread of the rot in the car even though incipient decay or spores were present when the fruit was packed. On the other hand if the trouble is one of the type with a wide host range, which is not typically a field disease but is apt to break out in transit or storage at

any time, then the responsibility rests with those whose duty it is to maintain proper sanitary conditions and an environment detrimental to the development of the disease while the product is in transit or storage.

*Dissemination of knowledge concerning diseases.* — The logical method of handling this problem is to educate the persons concerned, in the proper methods of combating these diseases and thus prevent the enormous losses rather than to continue the practice of attempting to collect damages from transportation companies and others who are supposed to be responsible for the losses sustained. This knowledge must be driven home to everyone concerned, from the grower to the retail dealer, and each must be made to assume his share of the responsibility for preventing these losses. In placing the responsibility for the occurrence of any particular type of disease or loss in storage or transit, the items discussed previously as to place of origin and contributing causes will be of great assistance.

The first party concerned is the grower. He is primarily responsible for the control of all those diseases which originate in the field. The grower probably has received more help from the plant pathologists in the past than anyone else concerned. A great deal of investigational work has been done on problems connected with field control of plant diseases and a great many bulletins have been published on this subject so that the grower has not been left without means of getting information dealing with his part of the problem. But not all growers have yet been reached with this information and not all who have been reached have acted upon the good advice. Much yet remains to be done in carrying the proper information on plant disease control to the grower and in inducing him to put this knowledge into use.

Then those who handle the product in harvesting, packing, stowing, and transporting have their part to play in disease prevention. First, the product should be in prime condition in every respect before harvesting; this involves some knowledge for which the pathologist is not directly responsible, but nevertheless knowledge the ignoring of which may later lead to disease in storage or transit. The processes of harvesting, packing, etc., involve so much handling that extreme care is necessary to avoid all the various mechanical injuries which are such important contributing causes to the fungous and bacterial rots, as previously discussed. There are so many people involved in the handling processes from the time the product leaves the field until it is placed on the market that their education in disease prevention assumes enormous proportions.

Cold storage and transportation companies have their part to play

in maintaining proper sanitation, refrigeration, heating and ventilation as precautionary measures against those diseases which may be prevented by these means.

It should be kept in mind that while a great deal of the information which should be imparted to these various agencies is already in the hands of plant pathologists, there are phases of the problem which need further research. This is especially true of the relation of environmental factors to diseases in storage and transit.

*Inspectors.* — The federal Food Products Inspection Law (24) enacted in 1917 enables the Secretary of Agriculture to investigate and certify to shippers concerning the soundness of food products when received at important markets such as he might designate. Under this law an extensive inspection service has grown up in the United States including all the larger cities. One of the duties of the inspectors is to determine the nature and amount of disease present in shipments of fruits and vegetables arriving at these large markets. Well trained inspectors at all these terminal points should be able to accomplish much in the way of determining responsibility for losses by disease, and to aid in the education of all concerned in methods of control. The ideal toward which all should look, of course, is complete and cordial coöperation between grower, packer, shipper, transportation agencies and wholesalers to the end that unnecessary losses from disease in perishable farm products stored or shipped to the markets shall be prevented.

#### REVIEW QUESTIONS

1. Mention the common types of plant disease troubles encountered in the storage, transportation and marketing of plant products.
2. Classify these troubles with reference to the time and place of origin.
3. What causes contribute to the occurrence of these diseases in storage, transportation or market?
4. Discuss the items included in Questions 2 and 3 as related to control.

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## PART II

### CHAPTER XIV

#### INTRODUCTORY — LABORATORY PROBLEMS

Part II of this book is devoted to a study of a number of plant diseases selected to illustrate the different types of plant maladies discussed in Part I under classification of diseases. (See Chapter III, page 26.) A study is made of one or more examples of each class of diseases listed in that outline, namely, those caused by slime molds; bacterial diseases; fungous diseases; those caused by parasitic seed plants; the nematode troubles; virus diseases; and the non-parasitic disorders. The diseases chosen for consideration have been classified in this manner because the writer feels that the most logical basis for the classification of diseases in a beginning course in plant pathology is that of the causal relations. The sequence here used is more or less arbitrary with respect to the larger groups in the classification, and within the parasitic group arrangement of the fungus diseases follows the generally accepted mycological order simply as a matter of convenience. It is not at all necessary for an instructor to follow the exact sequence in which the different diseases are discussed in the following pages.

Attention is called to the fact that this part of the book is both a text and a laboratory manual. Brief directions for laboratory study follow the text discussion of each disease. These outlines have been included for the benefit of those instructors who do not wish to write their own laboratory directions. As a preliminary preparation for the use of these outlines the following suggestions are given. By referring to these suggestions the instructor can modify or elaborate the laboratory exercise on any particular disease to suit his desires, or convenience. The list of review questions immediately following the discussion of each disease will be found helpful to the student whether or not the laboratory outlines are used.

#### Method of Study

In order that the student may have in mind a definite plan or model to follow in taking up the study of any specific disease the following tentative outline is offered. It is not intended that this outline shall

be followed rigidly in all cases, but it will serve as a guide and can be modified to suit the needs in any particular case, as the exercises following the different diseases illustrate.

1. **Laboratory study.** — The study of a disease in the laboratory may include the following points.

a. *Symptoms.* — The symptoms of the disease should be carefully observed and full notes should be taken. Note whether the symptoms are necrotic, hypoplastic, or hyperplastic. The student should constantly keep in mind the purpose of this study of symptoms, namely, to acquire ability to identify the disease wherever observed. The external signs of the disease, such as the fruiting structures of the causal organism, where such structures occur, are often of value in making accurate identification. Symptoms should also be studied under natural conditions in the field wherever that is possible.

b. *Pathological anatomy and histology.* — If available, sections of the diseased parts should be studied under the microscope in order to determine the effect of the disease upon the tissues and cells of the host. Interesting and valuable information concerning the nature of the disease can often be secured in this way.

c. *Morphology and life history of the causal organism.* — The study of a parasitic disease should always include a detailed study of the causal organism in all its forms and phases. It is particularly necessary to become familiar with whatever reproductive phases the organism may possess, and to understand its methods of perpetuation, dissemination and infection. A thorough knowledge of these phenomena is absolutely necessary in order that one may have a rational working basis for the control of the disease.

d. *Culture work.* — If time and equipment permit, it will be of interest to the student to make isolations and pure cultures of some of the disease-producing organisms. Inoculation experiments may also be performed. Instructions for carrying on culture work may be found in Chapter IV, and in the following references:

Duggar, B. M. *Fungous Diseases of Plants*, pp. 9-40.

Smith, E. F. *Bacterial Diseases of Plants*, pp. 76-131.

e. *Drawings.* — It is customary to require that certain drawings and notes be made in the laboratory. In general the drawings should illustrate two features: the symptoms of the disease and, in case of the parasitic diseases, the morphology of the causal organism. Whatever drawings are required should be carefully done. In general a few drawings well executed are to be preferred to many drawings carelessly done.

f. *Notes.* — The notes may be of two types. In some cases, perhaps, a brief outline of the main points of interest about a disease may be

sufficient. In other cases a longer and more detailed discourse may be desired. In either case some uniform system of note taking is desirable. The following outline is suggested as a model.

### Name of the Disease

Caused by (Insert name of causal agent).

1. Historical account.
2. Geographic distribution.
3. Hosts.
  - a. All species and varieties attacked.
  - b. Varietal susceptibility.
4. Symptoms.
5. Economic importance.
6. Etiology.
  - a. Morphology and life cycle of causal organism.  
(In case of parasitic diseases.)
    - (1) Vegetative phase.
    - (2) Reproductive phase.
      - (a) Perpetuation.
        - (1) Spore forms.
          - (a) Conidial stage.
          - (b) Perfect stage.
          - (c) Other spore forms.
        - (2) Sclerotia.
        - (3) Vegetative mycelium.
        - (4) Simple cell division (as in bacteria).
        - (5) Seeds (as in parasitic seed plants).
        - (6) Eggs (as in nematodes).
      - (b) Dissemination
        - (1) Time.
        - (2) Agencies.
      - (c) Infection.
        - (1) Time.
        - (2) Place.
        - (3) Manner of.
        - (4) Environmental factors.
          - (a) Favorable.
          - (b) Unfavorable.
  - b. Causal agents other than parasites.
7. Control measures.
8. References.

It will be noted that this outline provides for all possibilities as regards the cause of a disease. In writing up the notes on any particular disease the student, of course, will use only such parts of the outline under point 6, etiology, as fit the case in hand. For example, if he were discussing a non-parasitic disease, all points under morphology and life cycle of the causal organism would be omitted and a discussion of the causal agent, environmental or otherwise, would be substituted. Not all of the eight main points in this outline are of equal importance. Probably in most cases there are only three of these points that are of chief importance, namely, symptoms, cause and control. Such items as history, geography and economic importance may be of more or less interest but are not necessarily of paramount importance in gaining a fundamental knowledge of the cause and control of the disease. However, a judicious consideration of all the pertinent items in the outline will make a well balanced set of notes on any disease.

2. **Field Work.** — Wherever conditions permit it will be of great value to the student if the class can take several field trips during the term in order to observe and study plant diseases in their natural habitat. Any or all of the following topics may occupy the attention of the class on field trips.

*a. Identification.* — It is desirable that the student be able to identify, in all its forms, any disease studied, whenever he sees it. Extensive field work is valuable training for proficiency in identifying plant diseases.

*b. Observation of development.* — If possible, the student should make a series of observations extending over a considerable period of time in order to note different stages in the development of any particular disease. Otherwise he may be able to identify a disease in one stage of its development and fail to recognize it at another stage in its life history.

*c. Estimation of losses.* — When a disease has reached the stage at which the maximum damage it causes can be observed, the student should estimate percentages of loss due to disease. This will help to impress upon his mind the seriousness of plant diseases and the necessity for working out practical methods of control.

### Supplementary Reading

As each disease is studied it is desirable that the student read, in addition to the text, some of the better articles in the literature of the disease. By doing as much supplementary reading as time permits the student will not only gain additional knowledge not found in the text but will become acquainted with some of the sources of plant pathologi-

cal literature. The reading should be done, in so far as possible, before the notes on the exercise are completed, so that important facts recorded in the literature but not observed in the laboratory nor found in the text, can be included in the notes of the completed exercise. Some of the more important sources of information on plant diseases are listed below.

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- Annals of Botany. Oxford University Press. London.
- The Annals of Applied Biology. Cambridge University Press. London.
- Mycologia. Published for the New York Botanical Garden, by Lancaster Press, Inc., Lancaster, Pa.
- Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz. Verlag von Eugen Ulmer. Stuttgart, Germany.
- Bulletin of the Torrey Botanical Club. Lancaster, Pa.
- Memoirs of the Torrey Botanical Club. New York.

### 3. BULLETINS, CIRCULARS AND REPORTS

Among the chief sources of information on plant diseases are the numerous bulletins, circulars and reports published by the various agencies which carry on investigations and disseminate knowledge about plant pathological subjects. In the United States the most important of these agencies are (a) The Federal Department of Agriculture; (b) The state experiment stations; (c) The extension divisions of the state agricultural colleges; and (d) The state departments of Agriculture. In addition to these federal and state publications there are many valuable bulletins and reports published by endowed institutions such as The New York Botanical Garden, The Brooklyn Botanical Garden, The Boyce Thompson Institute for Plant Research, and The Crop Protection Institute. The proceedings of the various academies of science, state and otherwise, also contain valuable articles.

The governmental agencies of foreign countries also publish many bulletins and reports on plant diseases which constitute valuable reference material for workers in the United States. Many of these publications come from Canada, Australia and Great Britain.

### 4. BIBLIOGRAPHIES AND ABSTRACTS

Some of the most important lists of literature and abstracts dealing with plant pathological literature are found in the following publications: (a) Experiment Station Record; (b) Botanical Abstracts, now merged with (c) Biological Abstracts; (d) Agricultural Index; (e) Review of Applied Mycology (Issued by the Imperial Bureau of Mycology, Kew, England); and (f) Current Botanical Literature, formerly Current Author Entries (Issued by the Library, Bur. Pl. Ind., U. S. Dept Agr.). (Mimeographed.)

## CHAPTER XV

### DISEASES CAUSED BY SLIME MOLDS

The slime molds, technically known as Myxomycetes, constitute a group of organisms occupying a position very low in the scale of plant life, if they may be called plants. In some respects they resemble the fungi and it is this resemblance that has led to their inclusion in the plant kingdom. The character which distinguishes this group from other plant groups is the fact that the vegetative phase of a slime mold consists of a naked, multinucleate mass of protoplasm known as a plasmodium. This vegetative body resembles in consistency the white of an egg and may vary in color from practically colorless to varying shades of yellow, brown, orange, red, or violet. At maturity the plasmodium enters upon the fruiting stage by producing walled spores, as do the fungi. Furthermore, when fruiting, most of the slime molds produce some kind of a fruiting body in or on which the spores are borne. This gives them an added resemblance to the fungi. When the spores germinate they give rise to swarmspores which are amoeboid in nature. Both the swarmspores and the mature plasmodium to which they give rise have the power of locomotion by means of slow, creeping, amoeba-like movements. This characteristic of the slime molds in their vegetative stage has given rise to the question as to whether they are really plants or animals. Thus the term, mycetozoa, which is sometimes applied to this group, signifies this dual fungus-animal nature of the slime mold organisms. They are usually classed with the plants, however, because of the fact that they reproduce by means of spores and have other characters resembling the fungi.

The great majority of the slime molds are saprophytic and live upon decayed wood, leaves, or other vegetable detritus. Such species are usually of no economic importance, although occasionally a saprophytic form may grow up over a living plant, thus exerting a smothering effect upon it. On the other hand a very few species of this group of plants are true parasites and are the cause of serious diseases of agricultural plants. It is these pathogenic forms in which the student of plant pathology is chiefly interested. There are two slime mold diseases of cultivated plants which are particularly well known, namely, club-root of cabbage and other members of the mustard family, and powdery scab of potatoes.



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## Club-root of Crucifers

Caused by *Plasmodiophora brassicae* Wor.

In some truck-growing and gardening sections this disease frequently occurs on cabbages and other members of the mustard family. The disease affects the roots of susceptible plants and the deformities produced on these organs have given rise to the various names which have been applied to the malady such as "clubbing," "club-root," "finger and toe," "anbury," "kohlhernie" and "maladie digitore."

**Historical.** — The club-root disease of cabbage, turnips and other crucifers has been known in Europe for more than a century. It was mentioned as occurring in Scotland (6) as early as 1780 and again (1) in 1855 it was reported on turnips in that country. The first important work on the causal organism was done in Russia by Woronin (15), who published his completed studies in 1878. He established the fact that the disease is caused by a slime mold and worked out its general life history. In the United States the disease has apparently been known for half a century or more. In 1879 there appeared in the "Country Gentleman" two brief articles on club-root in cabbages (7, 8). There seemed to be some uncertainty in the minds of the authors of these articles as to whether the trouble reported was due to a fungus or to an insect, the cabbage maggot. It seems likely that both true club-root and the maggot injury were observed but that the two troubles were not clearly distinguished. In 1891 the disease was mentioned in the annual report of the Massachusetts Experiment Station (9). In 1892, Eycleshymer (6) published a good account of the disease in the United States, bringing together such information as was available up to that time in both this country and Europe. In 1913, Lutman (12) published the results of his investigations on the life history of the club-root organism and its effects on the host tissues. In 1918, Kunkel (10) gave further information on the manner of infection and migration in the tissues. The relation of soil temperature and moisture to infection was studied by Montieth (13) and the results published in 1924.

**Geographic distribution.** — The disease is apparently quite prevalent in Europe, especially in the countries of northern Europe, and the British

Isles. In the United States, club-root has been reported at different times from a large number of the states, especially in that section of the country east of the Mississippi river and north of the Ohio including the Atlantic seaboard states from Maryland northward. It has also been observed in the Pacific Northwest. The Plant Disease Bulletin (3) in summarizing the reports on this disease during the years 1903-1921, finds that during those years the disease had been reported at one time or another from the following states: Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, Pennsylvania, New Jersey, Delaware, Maryland, Virginia, West Virginia, North Carolina, Georgia, Alabama, Kentucky, Ohio, Indiana, Illinois, Michigan, Wisconsin, Minnesota, Iowa, North Dakota, Oregon, Washington and Alaska.

**Hosts and varietal susceptibility.** — The club-root organism confines its attacks to species and varieties belonging to the mustard family. Cabbage, turnips, cauliflower, Brussels sprouts, rutabagas and radishes are susceptible. Certain wild species of mustard also are attacked. Among these are the shepherd's purse, *Capsella bursa-pastoris*, and the hedge mustard, *Sisymbrium officinale*. Some differences in varietal susceptibility have been observed. In the United States, Cunningham (4) has carried out extensive investigations on the resistance of varieties of cabbage, radishes and turnips to the club-root pathogene. He found a wide variation in susceptibility among turnips. The White Swede, Sweet Russian and Sweet German were practically immune while the Southern Curled Turnip was very susceptible in his experiments. Cabbages showed a difference in varietal susceptibility but the variation was not as striking as among turnips. Resistant varieties of cabbages included Hollander, Stone Mason, Large Late Flat Dutch and Henderson's Early Summer, but these were by no means immune. Mammoth Rock Red, Dark Red Erfurt, American Savoy, All Seasons and Volga were among the more susceptible varieties. He found radishes also varying in susceptibility.

**Economic Importance.** — Few figures are available on the amount of loss due to club-root. Woronin (15), in 1876, stated that in the neighborhood of St. Petersburg, Russia, damage to the extent of \$225,000 occurred as a result of the club-root disease. In the United States, while the disease is known to occur in a majority of the states, it appears to be serious as a rule only in local areas here and there. In 1921 The Plant Disease Bulletin (3), reported a loss in Vermont of 1 to 3 per cent with the maximum amount found in any field placed at 10 per cent. In New York the percentage of loss was about the same as in Vermont. In 1923 an estimated reduction of 2.5 per cent in yield was reported (14) in

New York and Minnesota with a trace in Massachusetts, New Jersey, Maryland, Pennsylvania, Ohio, Indiana, Illinois, Michigan, Wisconsin, Iowa and North Dakota.

**Symptoms.** — The most characteristic symptoms of club-root are exhibited by the root system of the diseased plant, although the aërial parts of the plant also exhibit certain symptoms resulting from the failure of the diseased roots to function properly. In the great majority of cases the disease is manifested by a hyperplasia or overgrowth in the root system. Cunningham (4) recognizes six different types of

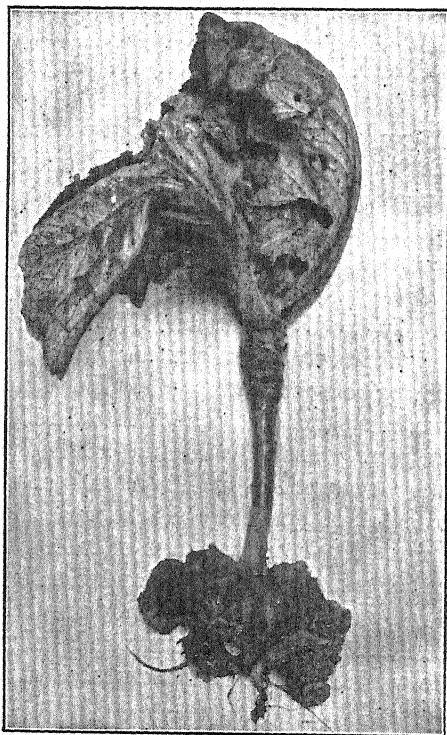


FIG. 10. — Cabbage plant affected with club-root, showing enlarged and deformed root system and the failure to develop a sound head. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

hypertrophy resulting in different species of crucifers attacked by the club-root organism. In all but one of these six types the effects consist of abnormal overgrowths of some kind. The first type consists of complete clubbing of both the main and lateral roots. This form is characteristic of the disease as found on cabbages and other varieties of *Brassica oleracea*. The whole root system ordinarily becomes swollen and deformed. In some cases it resembles a mass of fingers and toes. In other cases the main root becomes so much enlarged and clubbed as to assume the form of a very large, irregular, gnarled knot or tumor, three or four inches in diameter (Fig. 10). In the second type only the main root is enlarged, the laterals remaining healthy. This type is found on certain wild mustards. The third type is also found on wild mustards, and

clubs the lateral roots while the main root remains free from clubbing. Type four occurs on still other wild mustards and affects both main and lateral roots but healthy rootlets occur above the diseased parts. The fifth type is characteristic of the disease on turnips and radishes and

consists of tumors or lobulate swellings of the tap root (Fig. 11). The sixth type is found on certain varieties of radishes, and is characterized by cracks or fissures and darkened areas with true hypertrophy lacking.

The above-ground symptoms of the disease are various but all seriously diseased plants look sickly. Infected seedlings are stunted and sooner or later wilt, due to the fact that the root system can not furnish suffi-

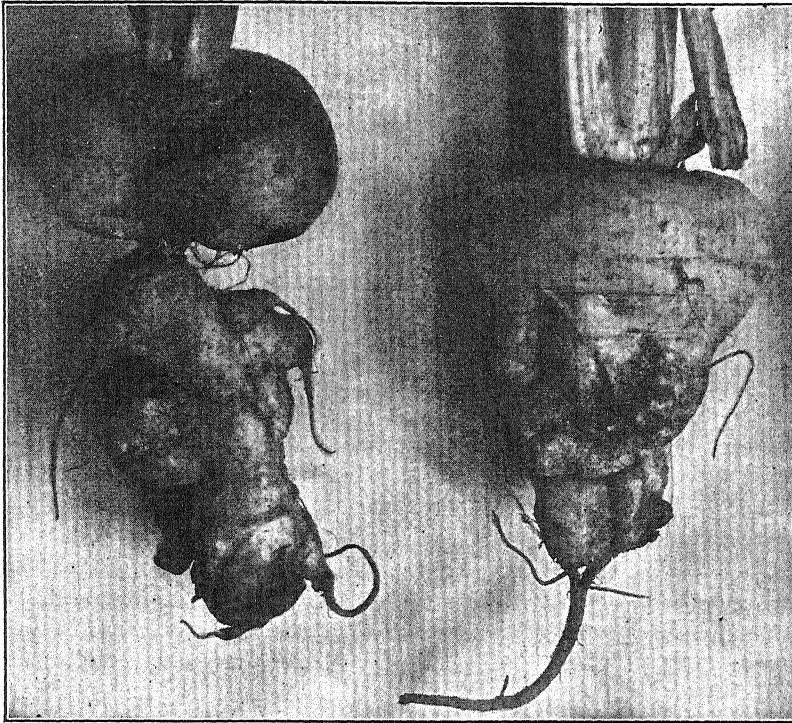


FIG. 11. — Turnips affected with club-root. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

cient water. Plants of this kind rarely form a head. If plants are older when infected they may head out but the heads are usually small and of inferior quality.

**Morphology and life cycle of the causal organism.** — The vegetative phase of this pathogene consists of a naked, amœboid mass of protoplasm which is sometimes referred to as a plasmodium. This amœba or plasmodium lives within the host cell and finally consumes the host protoplast and comes to occupy the entire lumen of the infested cell. At this stage a section of a diseased root shows many enlarged cells filled

with a dense granular protoplasmic mass which is light yellowish-brown in color (Fig. 12, A). When mature this plasmodium is multinucleate

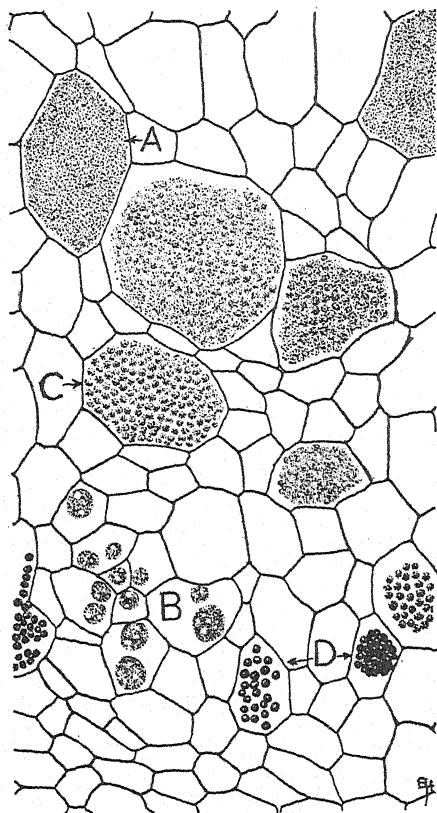


FIG. 12. — Section of a portion of a cabbage root showing various stages in the development of the club-root organism within the cells. A, plasmodium completely filling a cell; B, smaller amœboid forms which do not completely fill the cell cavity; C, a stage in spore formation; D, mature spores.

and spore production is effected by a process of division in which the plasmodium is separated into fragments in such manner that each nucleus with its surrounding portion of cytoplasm is inclosed by a wall and thus becomes a spore. These resting spores are spherical in shape and very small, averaging about  $3.3\mu$  in diameter, and a single diseased host cell sometimes contains a large number of these spores (Fig. 12, D).

*Perpetuation and dissemination.* — The organism is perpetuated by means of the resting spores which lie dormant in the diseased root or in the ground until the next season or until conditions are favorable for germination. The spores are liberated from the diseased roots when the tissues decay or when the roots are eaten by animals. Dissemination of the disease is accomplished by various agencies. Any diseased plants or parts of plants which are transported to new fields or localities undoubtedly carry the or-

ganism with them. This is especially true of seedlings taken from an infested seed bed. After the spores are liberated into the soil any agencies such as vehicles, farm implements and the feet of animals or man, which carry soil from one place to another, may carry spores along with the soil and deposit them in other places. Irrigation water or flood water may transport contaminated soil to clean fields. If diseased parts of plants are fed to animals without being cooked the

spores may be returned to the soil in the manure. It is doubtful if wind (2) is an important agency in scattering the disease. The motility of swarm spores is a negligible factor in dissemination since the spores can travel only very short distances under their own locomotion. The possibility of the transmission of the organism along with seed has never been demonstrated so far as the writer is aware, but it seems not improbable that spore-containing soil may come in contact with seed at harvesting time and that in this manner some spores might cling to the seed and be transported to new localities along with the seed.

*Spore germination.* — Chupp (2) perhaps gives the clearest description of spore germination. There is at first a decided swelling of the resting spore. Finally the pressure exerted splits the wall sufficiently to permit the living protoplast to ooze out. Motility begins when the protoplast is about half out of the old shell or spore wall. The activity increases until the living content of the spore has entirely escaped. The

freed spore now begins a rotary motion. When germination is complete the motile spore is termed a swarm spore because of its activities. It is

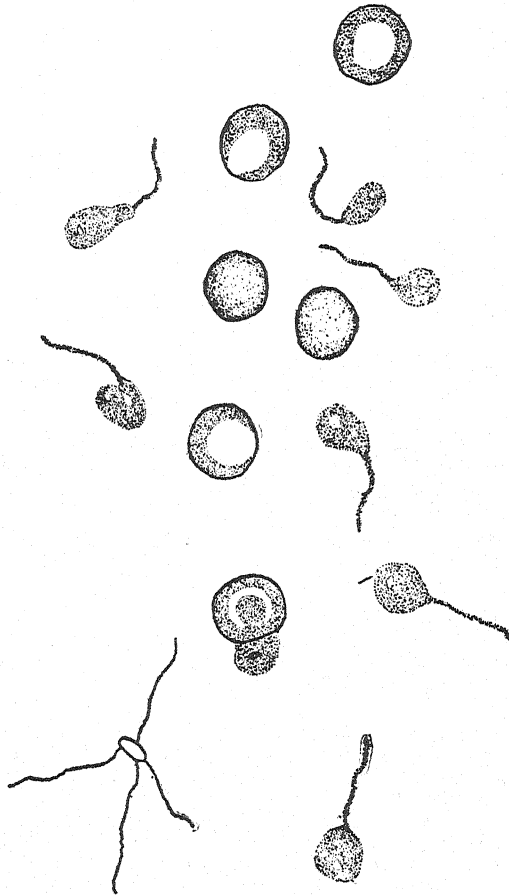


FIG. 13. — Germinating spores of *plasmodiophora brassicae*. Near the center are two ungerminated spores. At the top are two spores which have already germinated. Near the bottom is a germinating spore. The swarm spores which have emerged from the spores are each provided with a flagellum. At the lower left is a bacillus, an organism frequently found associated with *Plasmodiophora* in diseased roots. (After Chupp, Cornell Univ., Agr. Exp. Sta. Bul. 387.)

more or less pear-shaped and is provided with a flagellum at the smaller end of the body (Fig. 13).

*Infection.* — Penetration is effected by the swarm spores. The place and manner of penetration has been a subject for considerable investigation. Chupp (2) stated that his experimental work showed that infection takes place through the basal part of the root hairs. He believed that direct penetration into simple epidermal cells seldom, if ever, occurs. Later Kunkel (10) demonstrated that infection can take place through epidermal cells, other than root hairs, even on the stems of quite large plants and on the older roots.

*Distribution of the parasite within the host.* — It is generally agreed that after a swarm spore, or amoeba, enters the host cell it migrates to other cells by direct penetration of the cell wall. Thus if the amoebæ divide from time to time and some of the new individuals migrate to other cells, in a short time a considerable number of cells may become infected by the migrations starting from one original diseased cell. It is probably true also that some spread of the parasite within the host tissues is due to divisions of diseased cells, thus carrying amoebæ into the daughter cells. Kunkel (10) claims that most of the distribution of the parasite throughout the host tissues is due to migrations and that host cell divisions play a minor part in this distribution. At any rate, an examination of diseased tissue shows groups of diseased cells widely scattered throughout the parenchymatous tissues of the infested roots.

*Effect of environment.* — Various factors such as soil acidity and the temperature and moisture of the soil have been studied in their relation to infection and development of the club-root disease. For many years an acid soil has been considered as favorable for the development of this disease and control recommendations have included the application of lime to correct this condition. However, frequent failures of liming to control the disease have been observed and this has led to the study of other factors which might possibly enter into the problem. Montieth (13) has recently conducted investigations to determine the effects of variations in soil moisture and soil temperature on the club-root organism and the development of the disease. He found that the moisture content of the soil played an important part. The disease developed on plants grown in soil with a moisture content of 60 per cent or above but did not develop in soil held at 45 per cent or less of the water-holding capacity. He was of the opinion that this failure of the disease to develop on plants in soil with a low moisture content was due to lack of sufficient moisture for spore germination. The effect of temperature seemed to be indirect and related to its influence on host development. Club-root developed at temperatures ranging from 9° to



30° C. but was most pronounced at 20° to 25° C., which is the temperature at which normal root growth occurs most vigorously.

**Histology of the club.** — According to Kunkel (10) the cambium in young plants is invaded by the parasite soon after infection occurs. Subsequent to this invasion any new, undifferentiated cells laid down by the cambium are subject to infection. This interferes with the normal development of these cells into vascular elements as would happen in healthy plants. Consequently the water-carrying elements of the vascular bundle do not develop sufficiently to furnish an adequate supply of water to the leaves and upper part of the growing plant. As a result flagging or wilting ultimately occurs. When older plants are infected, the cambium and undifferentiated tissues are invaded but the older, woody parts of bundles are more or less immune from attack. The parenchymatous tissues of the medullary rays, however, are susceptible and may be invaded. This stimulates the ray cells to grow and divide. The overgrowth of the rays tends to split the bundles apart more or less and is responsible for certain types of distortion in diseased plants.

**Control.** — Scientifically devised control measures for any disease must necessarily be based on causal relations. Therefore, in order to make reasonable recommendations for the control of club-root one must consider all facts relating to the life history of the causal organism. The essential facts to be considered for this purpose are: (a) The organism is perpetuated by means of resting spores imbedded in the host tissue. (b) These spores are liberated into the soil upon the disintegration of the host tissue. (c) The spores may remain dormant in the soil for several years. (d) The disease is disseminated by any agencies which transport diseased plants or parts of plants, or contaminated soil. (e) The virulence of the organism is apparently favored by acid soils. (f) The parasite is favored by a high moisture content of the soil. (g) Some varieties are more susceptible than others. (h) The organism lives on weed hosts belonging to the mustard family.

Based upon the above facts the following recommendations seem logical: 1. Avoid transporting diseased plants into clean fields or localities. This includes seedlings from infested beds or nurseries, as well as diseased roots or parts of plants which may be returned to the land in compost or in any other form. 2. At harvesting time roots of diseased plants should not be left to decay in the soil. 3. Use a crop rotation of at least four years. 4. Keep down all weeds of the mustard family. 5. Avoid carrying soil by any means from a contaminated field to a clean field. 6. Neutralize acid soils with lime. 7. On irrigation projects avoid too free use of water. 8. Grow resistant varieties.



## LABORATORY STUDY OF CLUB-ROOT\*

A. **Symptoms.** — Examine specimens of diseased plants of different ages. Observe the abnormalities exhibited by both roots and aerial parts of the plants. How many different types of symptoms have been described as shown by different members of the mustard family when affected with this disease? (See text.) How many of these symptoms are shown by the specimens at hand? In the specimens of older plants note the dwarfing effect upon the head, or the failure to head at all. Make **drawings** to show all symptoms observed.

1. *Pathological anatomy and histology.* — Note the radial splitting of roots due to hyperplasia in the medullary rays. Examine sections of diseased roots with the microscope. Are all of the cells involved or is the disease confined to certain tissues? Is the enlargement of the root due to multiplication of cells, or to enlargement of individual cells, or both? Is the normal arrangement of cortical and vascular elements disturbed? Draw a portion of the section to illustrate these features.

B. **Morphology and life history of the causal organism.** — The organism is a slime mold, hence it has a vegetative phase which is amceba-like and a reproductive phase consisting of one spore form.

1. *Vegetative phase.* — This consists of a naked mass of protoplasm, a plasmodium, which lives within the host cell. Examine sections of diseased tissue and find cells filled with the plasmodia of the parasite. These can be recognized by their denser and more granular contents. Make a drawing to show the distribution of the infested cells.

2. *Reproductive phase.* — At maturity the plasmodium breaks up into a large number of small spores. A single cell of the host may contain a large number of spores. Find a spore-containing cell and draw much enlarged.

3. *Perpetuation, dissemination and infection.* — Consult the text and find out how the organism overwinters, how the spores are disseminated, their manner of germination, the place and manner of penetration of the host, and the way in which the organism spreads within the host.

C. **Notes.** — Write a complete account of this disease, including points observed in the laboratory as well as additional details obtained from reading the text and the appended references. For a suggestive model to follow in writing the notes, see the outline on page 152. This outline, especially under "etiology," contains items which are not applicable to this particular disease, therefore the student should select only those items which fit the case in hand.

## REVIEW QUESTIONS

1. Susceptibility to the club-root disease is confined to what family of plants?
2. Describe the symptoms of this disease. How many different types of symptoms are recognized by Cunningham on the various species of host plants?
3. Under what general class of symptoms would you place the symptoms of club-root as usually manifested on cabbages and turnips? (See Chapter III, page 23.)
4. To what class of organisms does the club-root pathogene belong? What characteristic of the vegetative phase in the life cycle of this class of organisms distinguishes them from the true fungi?

\* *Note.* — Before taking up the first laboratory study the student should read the discussion of laboratory work in Chapter XIV.

5. How is the disease perpetuated?
6. Describe the method of infection.
7. How does the organism spread in the host tissue after infection has occurred?
8. Describe the pathological histology of the diseased tissues.
9. What feature in the life history of the organism forms the basis for recommending crop rotation as a control measure?
10. What is the basis for recommending the application of lime to the soil as a control measure?
11. Discuss the relation of soil moisture to infection by the club-root organism.
12. Discuss the means of dissemination of the club-root pathogene.

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### Powdery Scab of Potatoes

Caused by *Spongospora subterranea* (Wallr.) Johnson

This potato disease has been known in Germany since 1841, and in England since 1846. The first reports of powdery scab in North America came from Canada in 1913 and from Maine in 1913 and 1914. The disease may have come originally from South America, the native home of the potato, since it was observed in Ecuador in 1891 and seemed to be well established there at that time. It attacks all underground parts of the potato plant, being most conspicuous on the tubers. The

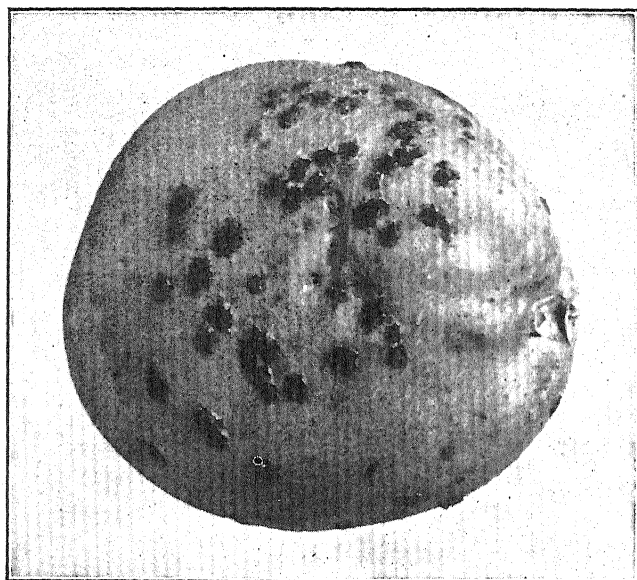


FIG. 14. — Powdery scab on potato tuber. (Photograph by F. D. Bailey, Ore. Agr. Exp. Sta.)

most typical and characteristic symptom is the "scab" which is really a pustule or blister which ranges from a millimeter or two up to four or five millimeters in diameter. At maturity the blister breaks open showing a powdery interior and leaving a fringe of broken skin around the margin of the pustule (Fig. 14). This typical pustule-like lesion serves to distinguish the powdery scab from the common scab of potato. (See under common scab in Chapter XX.)

The causal organism is a slime mold which penetrates the epidermis of the potato as a plasmodium and kills a small pocket of tissue. In the fruiting stage spores are formed which adhere in spongelike balls,

a single spore-ball containing several dozen spores. The powdery mass inside the mature pustule consists of many of these spore-balls together with the remains of broken down cell walls of the host tissue.

Powdery scab is very sensitive to climatic barriers so that it is not likely ever to become a serious menace over large areas of the United States. The disease thrives only under cool, moist conditions. Regions with heavy, cold, poorly drained soil offer the best conditions for the development of this disease while it practically always fails to develop in warm, light, well drained soils, even if repeatedly introduced. In localities where conditions are favorable for the development of the disease it may be successfully combated by the use of clean seed and crop rotation.

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## CHAPTER XVI

### PLANT DISEASES CAUSED BY BACTERIA

Some of the most widespread and harmful plant diseases known are caused by bacteria. It is only within the last fifty years, however, that pathologists have come to realize the truth of this statement. When it was first discovered that many of the diseases of man and of the lower animals were due to pathogenic bacteria, it was not then suspected that plants were also subject to attack by these microscopic organisms. Fungous diseases of plants have been known for a much longer time, but it was not until 1880 that a plant disease was proved to be caused by bacteria as distinguished from fungi. In 1880, Burrill definitely proved that bacterial germs cause fire-blight of pears and other pomeaceous fruits. Since the work of Burrill opened up the way, many other bacterial diseases of plants have been discovered and described. In 1920, Erwin F. Smith listed the families and genera of the spermatophytes in which bacterial diseases are known to occur. This list exceeds one hundred and fifty genera distributed through more than sixty families. In addition to these seed plants there are some cryptogams which are subject to attack by bacteria. A complete list of all known bacterial diseases of plants cannot be given here, but some of the more important ones include: fire-blight (*Bacillus amylovorus*), crown-gall (*Bacterium tumefaciens*), bean-blight (*Bacterium phaseoli*), cucurbit-wilt (*Bacillus tracheiphilus*), black-rot of crucifers (*Bacterium campestre*), soft-rot of carrot (*Bacillus carotovorus*), citrus canker (*Pseudomonas citri*), sweet corn wilt (*Aplanobacter stewarti*), black-leg of potato (*Bacillus strosepticus*), angular leaf-spot of cotton (*Bacterium malvacearum*), bacterial spot of stone fruits (*Bacterium pruni*), bacterial-gummosis of cherry (*Bacterium cerasi*), walnut-blight (*Bacterium juglandis*), brown-rot of Solanaceae (*Bacterium solanacearum*), and the olive tubercle (*Bacterium savastanoi*).

**Symptomatology of bacterial diseases.**—Bacteria cause several distinct types of symptoms in plants. The following are the most common forms of pathological effects found among the bacterial diseases of plants: (a) soft-rot, represented by the vegetable rot caused by *Bacillus carotovorus*; (b) the canker type, represented by fire-blight; (c) the leaf-spot, typified by the angular leaf-spot of cotton; (d) galls, as illustrated by the crown-gall and the olive tubercle; and (e) the vas-

cular wilt diseases, represented by the cucurbit-wilt and by the corn-wilt sometimes known as Stewart's disease of maize. A dwarfing is sometimes also produced but this is always in connection with some other symptom. An example of this is found in the potato blackleg disease where as a result of the rotting of the seed piece and the necrotic condition at the base of the stem the whole plant may be decidedly dwarfed before it is finally killed. Variations, modifications or combinations of any one or more of these symptoms sometimes occur in the same disease. For example, bean plants affected by the bacterial blight may manifest such a variety of symptoms as stem canker, leaf-spot, and wilt.

In the following pages a half dozen of the large number of known bacterial diseases are discussed briefly. They have been selected with a view, not only of presenting illustrations of the different types mentioned above, but also of representing a wide range of different hosts as well as a wide distribution throughout the United States. At least two of them, fire-blight and crown-gall, probably occur in every state in the Union, and the remainder, in addition to their wide distribution, represent other types of disease. In addition to those bacterial diseases which are discussed more or less in detail, a list of several others, with a few recent references on each, has been included at the end of the chapter, for the benefit of those who desire a more extensive knowledge of bacterial diseases.

### Fire-blight

Caused by *Bacillus amylovorus* (Burr.) Trev.

This very serious disease of pomaceous fruits has been known for a long time. Various names have been applied to it, probably the best known of which are pear-blight and fire-blight. Other names which have been applied to the disease, or phases of the disease, are twig-blight, blossom-blight, fruit-blight, collar-blight and blight-canker, depending upon the part of the host attacked; and apple-blight, quince-blight, etc., depending upon the kind of plant affected. The name "fire-blight" is now generally accepted and seems to be the most desirable designation for this disease, since it is descriptive of the most striking symptom, and all the other names suggested are more or less limited in their application.

**Historical.** — The first authentic report of this disease is probably that published by Denning (10) in 1794, in which he states that the disease was observed as early as about 1780 in the Hudson river high-

lands in New York State. It is probable that the disease originally occurred on the native crab apple and hawthorn and that when the early settlers began to grow orchards of susceptible fruits the disease spread to the cultivated varieties. From the time of this earliest record of the occurrence of fire-blight it seems to have spread throughout the United States until by 1880 it was well known and feared in the Central States, and in 1900 it had appeared on the Pacific Coast. The cause of the trouble remained a mystery until 1880 when Burrill (6) finally proved the disease to be due to a bacterial organism. In 1895 Waite (25) added materially to the accumulating knowledge concerning the nature and control of fire-blight. Since that time many investigators have contributed further information, especially on the manner of dissemination and methods of control.

**Geographic distribution.** — Fire-blight is widely distributed in North America but apparently had never escaped from this continent until quite recently. In 1919 the disease was discovered in New Zealand (7). This apparently is the first and only record of the occurrence of fire-blight outside of the North American continent. The disease is known to occur extensively in Canada. Within the United States, the disease is quite generally disseminated in most fruit growing sections where pears, apples and quinces, especially the former, are grown. The severity of the disease, however, does not seem to be uniform for all sections of the country. Some localities are comparatively free from serious outbreaks. The Willamette Valley, Oregon, is mentioned in this connection because the writer happens to be familiar with conditions here. While fire-blight has been observed in this valley at various times, it has always been in very small amounts and an epiphytotic has never been known in this region. Other parts of Oregon suffer severely from this disease. The exact cause for this freedom from fire-blight is not known but it is probably due to some ecological factor or combination of factors.

**Hosts and varietal susceptibility.** — The disease is largely confined to species of the pomaceous fruits, both cultivated and wild. The pear, apple and quince are the chief fruits attacked. Certain wild species of the Rose family are susceptible. These include the wild crab apple and species of hawthorn (*Crateagus*), service-berry (*Amelanchier*), and mountain ash (*Sorbus*). In addition to these pome fruits, some of the drupaceous or stone fruits are known to be slightly susceptible. Among the stone fruits, very light attacks of fire-blight have been reported upon the plums, cherries and apricots, but the damage done is insignificant in comparison with that suffered by the pome fruits.

Among the pears nearly all of the commercial varieties are more or

less susceptible but there is a great variation in degree of susceptibility among the different varieties. The Bartlett, Flemish, Clapp Favorite and LeConte are generally considered more susceptible to blight than such varieties as Kieffer, Duchess, Seckel and Winter Nelis. Other less well known varieties, such as Lincoln, Old Home and Estella have shown a marked degree of resistance. It should be remembered, however, that the performance of a given variety is not uniform under all conditions, so that a variety which shows a great degree of resistance at one time and under one set of conditions may be quite susceptible under other conditions. Thus the order of susceptibility among varieties may change to a certain degree with changing conditions. Recently certain Chinese species of pears which have been imported into this country and tested have shown a remarkable degree of resistance to blight. These will be discussed more in detail below, under Control.

**Economic importance.** — Fire-blight is one of the most serious of all diseases attacking pome fruits, particularly pears. In some pear growing sections it is the most dreaded of all diseases and in many cases threatens to eliminate the pear growing industry. Losses from this disease in California in 1923 were estimated at \$2,000,000. Pears are not a commercial crop in Indiana, because of blight. In Florida, fire-blight is reported to be responsible for the abandonment of pear growing in many sections (1). Blight is considered the limiting factor in many other states, including Kentucky, North Carolina, Gulf States, Arkansas, New Mexico, Arizona, Utah and Idaho. In Oregon, particularly in the Rogue river valley, blight is a menace to pear-growing.

The damage done to apple orchards is extensive in some cases, but on the whole the disease is not so disastrous to apples as to pears. In some apple-growing sections a few blight-infested pear trees in close proximity to apple orchards have proved to be bad centers of infection for the apples. When these diseased pear trees have been removed the apples no longer suffer seriously from blight (1).

The nature of the damage done by fire-blight is chiefly that of girdling and killing twigs and branches, and even whole trees.

**Symptoms.** — All parts of the host show the effect of the disease. It most frequently appears as a blighting of the twigs, but blossoms, leaves, branches, trunks and roots may be invaded by the organism.

*On blossoms.* — The first new infections in the spring usually take place in the blossoms. Within two or three weeks after blossoming, the dead, brown, or blackened tufts of blossoms and leaves become evident. It commonly happens that if a blossom is inoculated the organisms work back at least as far as the tip of the spur, so that all blossoms and leaves in that cluster are killed.



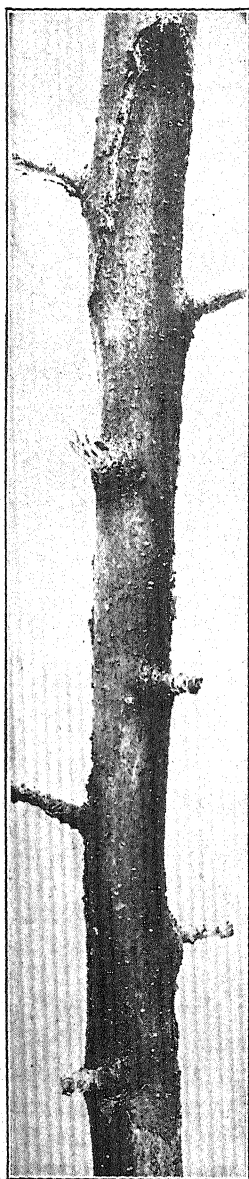


FIG. 15. — Fire-blight canker on apple tree. (Photograph by Jackson, Ore. Agr. Exp. Sta.)

*On twigs.* — Frequently the bacteria in a diseased blossom do not stop spreading at the spur but continue into the twig at the base of the spur, killing it. Also infection may take place in the tip of the young growing shoot, probably from insect punctures or bites. In any case, the characteristic symptoms on blighted twigs are manifested by the dead, brown, or blackened leaves which cling tenaciously to the twig for a long time after death.

*On leaves.* — Although as a rule the dead leaves found in connection with fire-blight injury result from the death of twigs and spurs, yet there is some evidence (12, 14) that leaves may sometimes be invaded directly by bacteria which gain entrance through the stomata or perhaps through wounds on some part of the leaf. In case of this type of leaf invasion, the brown areas usually extend inward from the margin for a variable distance, sometimes entirely to the midrib. This type of leaf invasion should be distinguished from that in which the bacteria from an infected twig travel out through the petiole and along the midrib into the leaf. In either case there is apt to be more or less exudation of ooze containing the germs of the disease.

*Branches.* — Whether the original infection starts on a blossom, a leaf, or at the tip of a succulent shoot, seems to make no difference in the subsequent spread of the disease. Whatever the point of entrance, if conditions are right, the disease may spread indefinitely down into the larger branches, either girdling them or forming large elongated or irregular dead areas along one side of the branch. The bacteria migrate mostly in the cortical region and the invaded bark at first shows a more or less water-soaked appearance on the surface, later turning darker in color, especially in the case of the pear. In the early stages there is not a sharp line of demar-

cation between healthy and diseased bark, but after the disease stops spreading the killed area gradually dries out, so that finally the canker becomes bounded by a crack between the dead and living areas (Fig. 15).

*Trunk and roots.* — The disease may continue spreading down from the branches into the trunk and even into the roots, or new infections may take place in wounds or in suckers or watersprouts starting out from the trunk or roots. Sometimes the trunk may be partially or entirely girdled near the ground. This type of injury is sometimes



FIG. 16. — Pear fruit showing effect of invasion by the fire-blight organism. The pearly beads on the surface are drops of exudate containing large numbers of bacteria. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

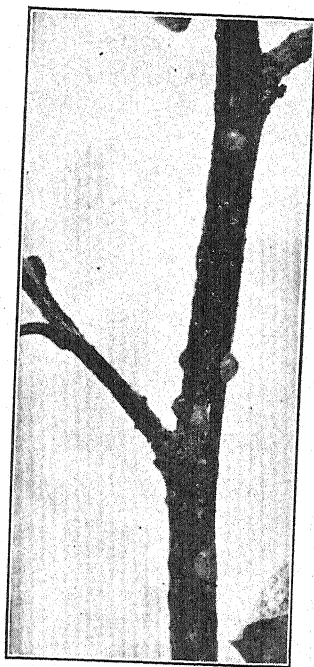


FIG. 17. — Blighted twig of quince showing drops of bacterial exudate. (Photograph by F. D. Bailey, Ore. Agr. Exp. Sta.)

called "collar-blight." These trunk and root infections often result in the death of the entire tree.

*Fruits.* — Frequently fruits become infected. This may happen at any stage in their development. The germs either travel out into the

fruit from a diseased twig, or the fruit may be inoculated directly through the activities of insects (Fig. 16).

*Ooze.* — Any diseased part when active and sappy is likely to give rise to an exudate of sticky sap which usually shows as droplets on the surface of the bark, leaves or fruit. This exudate is at first of a milky or opalescent color, later turning darker. The ooze always contains numerous active germs and is the most common natural source of inoculum (Figs. 16, 17).

**Morphology and life cycle of causal organism.** — The bacterial organism causing fire-blight is a flagellate rod belonging to the genus *Bacillus*. It perpetuates itself by simple cell division as is typical of the bacteria in general. It winters, in cyst-like masses (17), only in diseased bark of its host plants, especially around the margins of the larger killed areas. These overwintering centers are known as hold-over cankers. The cankers on the larger branches, trunk and roots are especially favorable for the overwintering of the organism.

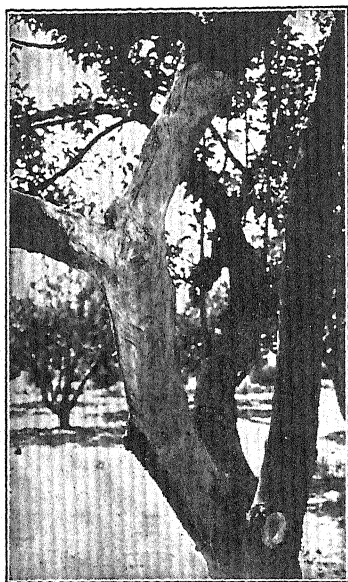


FIG. 18. — Large fire-blight canker on apple tree. The diseased bark has been shaved off, showing the extent of the invasion. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

*Dissemination.* — Insects are probably the most important agents of dissemination. When the sap begins to rise and the tree resumes activity in the spring, the dormant bacteria become active, and begin to spread into healthy bark surrounding the old canker. Under these conditions the cankers exude a sticky, sappy ooze which contains innumerable bacteria and which is attractive to insects. Many different kinds of insects have been named as probable agents. Among these are bees, wasps, aphids and the tarnished plant bug. Honey bees have been blamed for a great deal of fire-blight dissemination at blossoming time. Gossard and Walton (11) have shown that fire-blight bacteria will live in honey for at least seventy-

two hours and produce blight if twigs are inoculated with this honey. They also claim that this organism is probably carried in the pollen baskets of bees. However, they state that any harm that bees may do

by disseminating fire-blight germs is more than compensated for by the pollination accomplished by the bees. The claim is made that blossoms are not likely to be inoculated seventy-two hours after pollination and are immune to fire-blight organisms one hundred and forty-four hours after pollination has occurred.

Rain is undoubtedly responsible for a certain amount of dissemination through the spattering of ooze about the tree.

Man certainly disseminates the disease widely in his pruning and cultural operations unless precautions are taken to prevent it. The organisms are so numerous in the diseased bark that any tool which is used in cutting out diseased parts is very apt to become smeared with the germs and when the next cut into healthy tissue is made the organism is introduced.

*Infection.* — Inoculation takes place readily in blossoms whenever the organisms are carried there by insects or other agencies. The nectaries and stigmas are probably the points of infection. Inoculation also apparently occurs in young shoots through the agency of sucking or biting insects. Wounds of any sort offer a point of entrance, provided the bacteria find their way to them. Direct invasion of leaves may occur, probably through stomata or water-pores, or through punctures or abrasions of the leaf epidermis (14). Infection and spread are facilitated by an increased degree of succulence in the host. Weather and other conditions which keep the trees in a rapidly growing and excessively sappy condition favor infection and the rapid migration of the bacteria in the tissues.

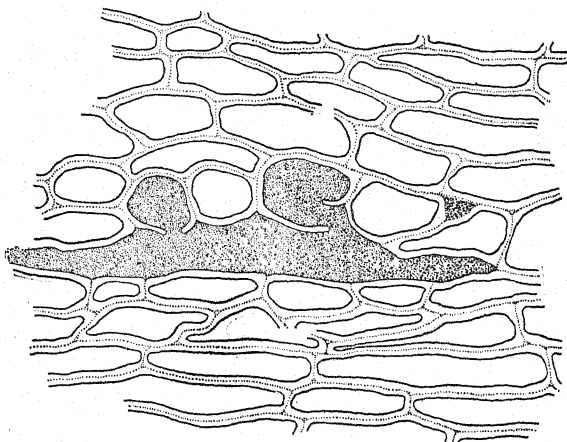


FIG. 19. — Section of diseased pear bark showing a group of the fire-blight bacteria in the intercellular spaces and in the space where cells have separated. They are also shown within the lumen of certain cells the walls of which are ruptured.

*Migration in the host tissue.* — The method by which the fire-blight bacillus migrates in the tissues of the host has been the subject of con-

siderable speculation. It has been generally supposed that the bacteria move along in the intercellular spaces in some way but the manner of locomotion has not been clearly demonstrated. It has also been stated that the organism sometimes occurs within cells, the walls of which are apparently intact. In the latter case, the explanation of the manner of entrance has been highly conjectural. The most recent contribution to our knowledge of this subject has been made by Nixon (17). He states that during the period of migration, the fire-blight bacteria occur imbedded in masses of jelly-like material termed zoöglœæ. These masses progress through the intercellular spaces by pushing out pseudopod-like projections between the cells. Apparently, the middle lamella is broken down in places, since the cells separate extensively, forming large schizogenous cavities. During the early stages of invasion, the organism cannot be found within the cells. Later, the cell walls and also the protoplasts are dissolved, thus forming large lysigenous cavities. During this phase the organism is found inside the host cells, presumably entering through the partially dissolved walls.

**Control.** — Fire-blight is very difficult to control. Up to the present time no entirely satisfactory method has been devised for combating it. There are five possible lines of attack, all of which may be beneficial to a greater or lesser extent. These are: (a) surgery, (b) sanitation, (c) insect control, (d) immunization, and (e) cultural practices.

**Surgery.** — The disease can be held in check to a certain extent by cutting out all infected parts as soon as they are discovered and thus lessening the sources of further infection. In case of extensive infestation this entails an enormous amount of work and requires keen powers of observation in locating all diseased parts and extreme care in cutting out all these parts without unnecessarily spreading the disease further. In some severely infested sections it has been only by eternal vigilance in watching for and cutting out every diseased twig or branch and every canker on trunk and roots that valuable orchards have been saved from destruction. In the cutting out process it is absolutely necessary that both tools and wounds be thoroughly disinfected. It is also necessary to cut out every bit of diseased tissue. When cutting through diseased parts the tools become contaminated with the germs and if these are not killed by an effective germicide before the final cut is made the wound will be re-inoculated and the disease will continue to spread. Several different chemicals have been used in the past for disinfecting tools and wounds, the best known of which is probably corrosive sublimate (bichloride of mercury). However, it has been discovered in recent years that this germicide is not effective on fresh wounds and a better disinfectant has been found (20). This consists of 1 gram of bi-

chloride of mercury and 1 gram of cyanide of mercury dissolved in 500 cc. of water.

The disinfectant is carried in a bottle and a sponge is used in applying it to the wound or tool every time a twig is cut off or a canker cut out. In cutting out cankers the procedure varies somewhat, depending upon the age of the canker. In young cankers the bacteria migrate in the outer spongy bark and this type can be treated by shaving off the outer bark without destroying the cambium and then applying the disinfectant to the exposed surface (9). After several weeks, however, the bacteria penetrate to the cambium and then in treating it is necessary to shave off all bark down to the wood. For disinfecting after cutting out the younger, shallow canker it is recommended (8) that glycerine be added to the above disinfectant. This prevents the rapid drying out of the exposed surface and thus allows more rapid healing of the wound.

*Sanitation.* — In cutting out blighted parts all prunings should be burned immediately. It has been shown (15) that the bacteria may live for several weeks in prunings left on the ground in shaded or moist places and that there may be exudation of germs from these twigs and branches in wet weather.

*Insect control.* — Since insects are largely responsible for the dissemination of fire-blight it has been suggested that the use of insecticides and insect repellants might prove effective. This has been tried with a certain amount of success against such insects as thrips and aphids, but it seems doubtful if this method will ever be widely and successfully used.

*Destruction of "weed" hosts.* — In regions where the hawthorn (*crataegus*), service-berry (*Amelanchier*) and wild crab apple occur, these species act as overwintering hosts for the fire-blight organism and thus may serve as a source of infection for the pome fruits. All such "weed" hosts should be eradicated from the neighborhood of pear and apple orchards. None of these "weed" hosts should be left growing within a distance of a mile from the orchard.

*Cultural practices.* — Since an increased degree of succulence in the host seems to increase the probability of infection and rapid spread, any cultural practices which tend to inhibit too vigorous growth of the trees during the summer will, in general, be considered desirable from the standpoint of controlling fire-blight. In irrigated sections this can be effected by limiting the water supply, and in any section a curtailment of cultivation may be beneficial in this respect. Pruning practices which tend to stimulate abundant production of new, succulent wood should also be avoided. In any case a balance will have to be struck

which will allow the production of a good crop and at the same time reduce the danger from fire-blight as much as possible.

*Resistant varieties.* — Reimer (19, 21) has secured several species of Chinese pears, notably *Pyrus ussuriensis* and *P. calleryana* which are practically immune, and is now using these in grafting and budding, by which means young nursery stock practically immune to blight is produced. These young trees are then used for top working to the desirable but susceptible varieties. In this way, it is hoped ultimately to supply the country with pear trees which will never be killed outright by fire-blight, although the twigs of the susceptible tops may still blight.

#### LABORATORY STUDY OF FIRE-BLIGHT

**A. Symptoms.** — All parts of the susceptible plant, including blossoms, leaves, fruits, twigs, branches, trunk and roots, are subject to attack by the fire-blight organism. Examine diseased specimens of all parts available. Note especially the blighted twigs, to which the dead leaves cling tenaciously, the invaded fruits, and the cankered areas on the stems. Can you trace the limits of the killed areas on branch, trunk or root? Note drops of exudate on various invaded parts. On what parts of the host and under what conditions may this exudate appear? Make sketches to illustrate the various symptoms.

**B. Relation of parasite to host tissues.** — Examine, under the microscope, sections of both healthy and diseased pear twigs. In the sections of diseased twigs you should be able to locate groups of bacteria in the bark tissue. Are the bacteria between the cells, inside the cells, or both? Can you detect any striking effect of their presence upon the host cells? Compare with the healthy twigs. Can you detect bacteria in any other than the cortex tissue? Draw a part of the section to show location of the bacteria.

**C. Morphology and life history of the causal organism.** — Examine pure cultures of the bacteria and describe the cultural characteristics. Make mounts and examine the organism under the microscope. Examine stained mounts of the bacteria with the oil emersion lens. Has the organism cilia? Draw. If mounts are not available, observe illustrations in the literature.

**1. Perpetuation, dissemination and infection.** — Find out where the organism overwinters. What part does the exudate play in the life cycle? Is the exudate more concerned with perpetuation or with dissemination? What are the disseminating agents? What conditions favor the spread of the disease? Look up these points in the text and in the list of references following.

**D. Notes.** — Write complete notes embodying all information obtained from any source upon this disease.

#### REVIEW QUESTIONS

1. Name all parts of the plant which may be attacked by fire-blight and describe the effects of the disease on each part.
2. Describe the manner of perpetuation, dissemination and infection.
3. What have climatic, seasonal and weather conditions to do with the spread and seriousness of fire-blight?
4. Discuss the relation of insects to the dissemination of fire-blight. (See references 11 and 24.)

5. How does the fire-blight organism migrate within the host tissue? (See references 4 and 17.)
6. Discuss the problem of cutting out fire-blight without spreading the disease. (See references 8, 9, 20.)
7. Discuss the problem of combating this disease by means of resistant varieties. (See reference 21.)
8. How long will the fire-blight bacteria remain alive in twigs after they have been cut off? (See reference 15.)

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### Crown-gall

Caused by *Bacterium tumefaciens* Smith and Townsend

Crown-gall is a disease which is well known to fruit growers in many parts of the world. It has been given a variety of names such as crown-knot, woolly-knot, root-knot, tuberculosis, root-tumor, black-knot, hairy-root and plant cancer, in addition to the term crown-gall, by which designation it is usually known in the United States. The term "crown-gall" probably originated from the fact that the swellings or galls commonly occur at or near the so-called crown of the plant near the surface of the ground. As will be noted later the majority of galls in nursery stock occur near the point of union of stock and scion.

**Historical.** — The disease apparently first came into prominence as a malady of the grape in Europe. The first account of the disease on the vine was given by Fabre and Dunal (4) in 1853. Thus it has been known in Europe for at least three quarters of a century. A form of the disease called "Grind" was described in Germany by Dornfield (3) in 1859. Many other investigators in France, Germany and Italy have described the trouble on the vine and also on other plants including fruit trees, forest trees and shrubs. Cavara (2) in 1897 seems to have been the first to isolate the organism from vine tumors and make successful inoculations. Previous to that time many speculations as to the cause of the trouble had been indulged in by the various workers but nothing definite was known about it. In the United States there is little mention of crown-gall previous to about 1890. Galloway (5) described a disease of grape vines which he called black knot, in 1889. One of the earliest workers in this country was E. F. Smith who later became the leader in crown-gall investigations. For many years the work here was descriptive with unsuccessful attempts to determine the cause. Beginning in 1904 and extending over a period of some ten or

fifteen years, Smith and his associates (27, 28, 30, 31, 32) conducted a series of intensive researches which not only successfully demonstrated the cause to be a bacterium but also brought out many other interesting facts concerning the nature of crown-gall and its host range. More recently Riker (16, 17, 18, 19, 20), and Riker and Keitt (21, 22, 23), have added materially to our knowledge of this disease.

**Geographical distribution.** — The distribution of crown-gall is apparently world-wide. It has been reported from many of the countries of Europe, especially the grape-growing regions. It is known in South Africa on the grape, and large galls which may be this disease occur in that country on a variety of trees including apple, peach, poplar and willow. Reports come also from Australia, New Zealand and Asia. In North America it occurs in Canada, the United States and Mexico. It is now known all over the United States, being reported from practically every state in the Union.

**Hosts.** — Probably the most important family attacked by the crown-gall organism is the rose family, including apple, pear, peach and raspberry. Smith (29) lists about 40 species in 18 different families which he found cross-inoculable with the crown-gall organism. These include potato, tomato, tobacco, daisy, cabbage, cauliflower, turnip, radish, beet, carrot, grape, oleander, clover, almond, carnation, hop, Coleus, Citrus, Impatiens, Opuntia, walnut, poplar, mango, castor bean, cassava, Fuchsia, Salvia and sunflower. Probably many others are susceptible.

There seems to be a great variation in varietal susceptibility to crown-gall although this variation is not always uniform and may depend to a certain extent on external factors, mainly ecological. In California (7) varieties of grapes (*Vitis vinifera*) showing great susceptibility are Mission, Muscat, Tokay and Malaga. Some resistant varieties of the species are Rupestris, St. George, Sweetwater, Seedless Sultana, Carignane and Grenache. Among apples the Ben Davis, Early Harvest, Yellow Transparent, Wealthy, Grimes, Northern Spy, Oldenburg, Wolf River, Gano and Rome Beauty are said to be most susceptible. In Iowa (11) experiments indicate that Wealthy is more susceptible than Jonathan. In some localities Red June, McBride and Delicious are listed as less susceptible varieties.

**Symptoms.** — On tree hosts the galls are usually confined to the underground parts, either at the base of the trunk or on the roots near their junction with the trunk of the tree. They are seldom found far down on the roots. On grapes and the bramble fruits the galls are frequently aerial. A very high percentage of crown-gall occurs on nursery stock, where it appears on the scion just above the point of union with the stock. The usual symptoms have been classified as (a)

galls and (b) hairy-roots. The young galls first appear as small, slightly greenish or flesh-colored, soft, spongy or fleshy growths. As they

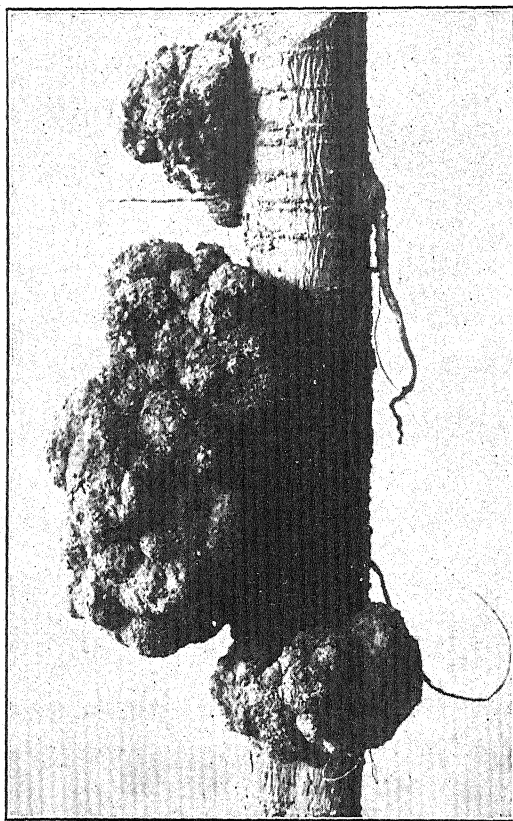


FIG. 20. — Typical crown-gall on peach root of prune tree. (After Barss, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

mature they become darker in color and enlarge, frequently becoming several inches in diameter, with irregularly roughened surface, and are usually hard or woody in texture (Fig. 20, 21). On vines and cane fruits the galls are frequently elongated in a longitudinal direction along the stem and may become many inches long, not infrequently a foot or more (Fig. 22). Some galls remain soft throughout their existence. Smith (32) says that whether or not a gall shall be of the soft or the hard type depends upon which meristematic cells receive the initial impulse. If the mother cells of parenchymatous tissues are first invaded they are stimulated to produce more parenchyma

cells, with the result that a soft gall is produced. On the other hand, if the inoculum reaches the cells which give rise to the woody tissues a hard gall will result. The hairy-root form is characterized by a dense growth of small fibrous roots usually arising from a slight gall-like swelling on the base of the stem or on the larger roots (Fig. 23).

Experimental inoculation work with the crown-gall organism has produced many peculiar or freakish growths on plants (9, 27). Frequently various plant organs, as leaves, flowers or roots, in more or less abnormal condition, may grow out from gall tissue or arise directly at the points inoculated.

*Doubtful cases.* — For many years a peculiar burl-like growth frequently occurring on branches of apple trees has been thought by many observers to be an aërial form of crown-gall. Recently evidence has been brought forth indicating that these tumors are not due to the

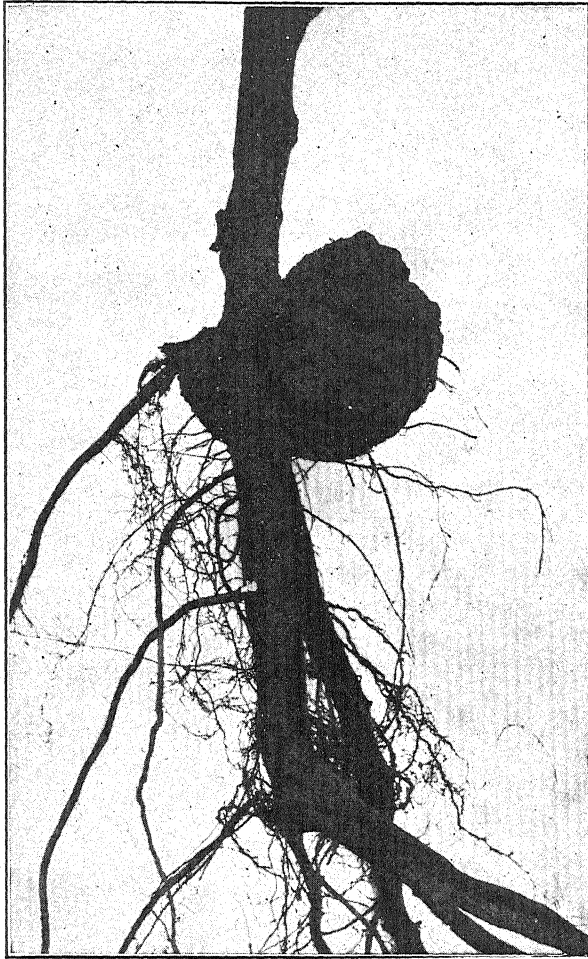


FIG. 21. — Crown-gall on apple tree. (Photograph from files of the Ore. Agr. Exp. Sta.)

crown-gall organism (1). Doubt has also been cast upon the identity of many gall-like growths which appear at the union in grafted nursery stock (21, 22, 23). Many swellings which formerly passed as crown-gall are now known to be abnormal callus formations (Fig. 24) and not

due to *B. tumefaciens*. Also it has been discovered that not all hairy-root symptoms are due to true crown-gall, although it is certain that some hairy-root growths are due to infection by the organism of crown-gall. The woolly-knot type is infectious, while the type in which fine fibrous



FIG. 22. — Crown-gall on grape vine. (After Barss, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

roots arise in clusters from the tap root is probably not pathogenic (13, 14).

**Economic importance.** — Crown-gall has long been considered a serious disease of both tree fruits and small fruits. It is especially prevalent in nurseries, although in the light of recent investigations, it seems that other abnormalities have been confused with true crown-gall. This discovery of course lowers the percentage of the genuine disease in comparison with former estimates. It is very difficult to accurately

estimate the damage caused by crown-gall because the trees are not killed outright and in many cases heavily galled trees may live for years without showing any very serious effects.

On the other hand, it has been shown that the presence of galls, especially at the graft union, interferes to a greater or lesser extent with the passage of water up the stem. In a set of experiments conducted at the

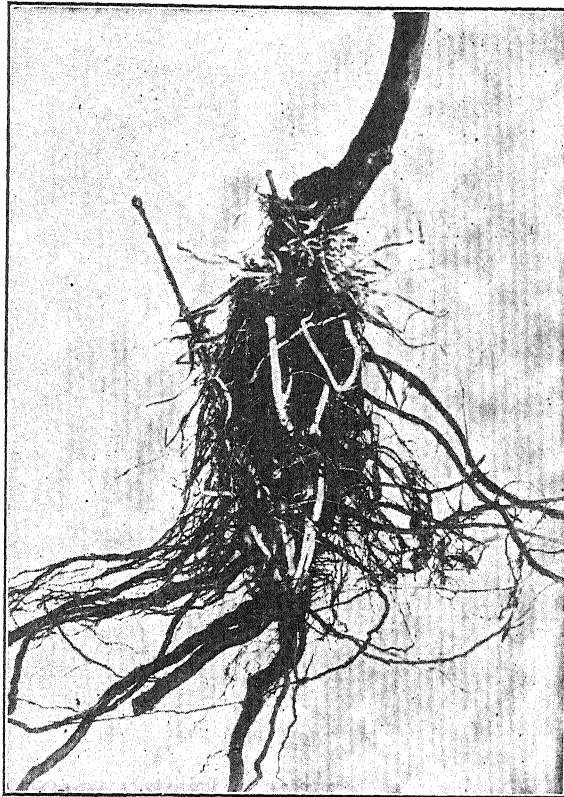


FIG. 23. — Hairy-root on young apple tree. (Photograph by Barss, Ore. Agr. Exp. Sta.)

Iowa Experiment Station (12) it was determined by a specially constructed piece of apparatus that the rate of water flow through stems galled at the graft union was considerably reduced over that in healthy trees. Two-year-old apple trees were used and 100 galled and 100 healthy trees selected for the trial. The average reduction in the rate of water flow through the diseased stems was 30 per cent in this experiment. Continuation of this experimental work (15) in later trials

showed a water flow reduction of 69.7 per cent in Wealthy, 21.7 per cent in Salome, and 47.2 in Jonathan apple trees. Such a reduction must undoubtedly have a harmful effect upon the tree, especially under conditions of high transpiration and scant water supply.

Several attempts have been made to secure more accurate information on the actual damage resulting from this disease over a period of years.

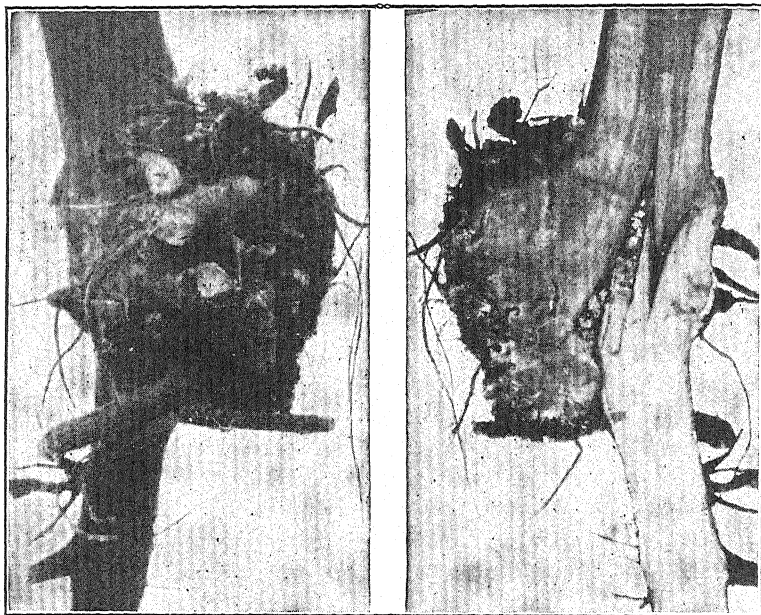


FIG. 24. — Surface and sectional views of wound overgrowths at the union of three-year-old grafted apple trees. Left, surface view of the union; right, section of the enlargement shown at left. The lower tip of the cion extended beyond the cambium on the stock and wound overgrowth developed. (After Riker and Keitt.)

Typically galled or hairy-root nursery stock has been planted out under actual orchard conditions and allowed to grow for several years under observation to determine the nature and amount of injury resulting. In Montana (34) an orchard of 240 apple trees, half healthy and half diseased, was set out in 1910 and observed through eight growing seasons. The general conclusions drawn from this experiment were: (a) Apple trees affected with crown-gall when planted in the orchard will seldom make as good trees as healthy stock, although no definite predictions can be made in specific cases. Some trees may apparently be as good as those which were healthy at transplanting, while others may be seriously dwarfed. (b) The roots are dwarfed more than the parts above



ground. (c) If hairy-root develops at the expense of strong anchor roots, it is very important, but if the root system is otherwise well developed it will do little damage. (d) Galls located at the crown are more harmful than those on the lateral roots. (e) The damage to trees results from interference with food conduction. Galls also provide an easy means of access for other diseases. (f) In this experiment the Northwestern Greening variety showed the least injury, while Hibernial and Patten were more severely dwarfed. A similar experiment was begun in Iowa (11) in 1912. Only two varieties, Jonathan and Wealthy, were used. The general conclusions drawn from the Iowa experiment follow: (a) Jonathan is more resistant to gall than Wealthy. (b) galled trees grow much more slowly than healthy ones. Crown-gall greatly retards the growth of young trees, resulting in a reduction in the rate of increase of trunk diameter, and also in less twig growth. (c) Twig measurement seems to be a more accurate index of the effect of crown-gall than trunk diameter measurements. (d) Fruit spurs are stimulated to earlier development on badly galled than on normal trees. (e) Galls occurring on the union or stock are more harmful than those on the secondary roots.

The actual per cent of loss from crown-gall is very difficult to estimate in the orchard but may be more accurately determined in nursery stock. The Plant Disease Reporter, Supplement 20, reports the following items for the year 1921. In Wisconsin a loss of 15 per cent in nursery stock was reported. In Kansas 25 per cent infected stock occurred in nurseries. Out of 60,000 trees shipped into Maryland from other states 20,000 were destroyed because they were badly galled. The Reporter, Supplement 28, estimates a loss of 25 per cent in Iowa nurseries in 1922. Supplement 39 of the same publication reports 50 per cent of nursery stock affected in Tennessee in 1924. In the same year a maximum of 90 per cent was reported for certain nurseries in Mississippi, and 50 per cent in New Mexico.

Crown-gall is very prevalent on grapes and undoubtedly does a great deal of damage to this crop. In the early history of the disease in Europe it is credited with the destruction of whole vineyards. In the United States considerable damage results on European varieties, the American varieties being more resistant. Raspberries also suffer severely from crown-gall. In some localities crown-gall is said to be the limiting factor in the culture of cane fruits, noticeably so in parts of Wisconsin, Illinois and Minnesota.

**The causal organism.** — *Bacterium tumefaciens* is a rod-shaped, motile, polar flagellate bacterium, multiplying by fission. It grows on agar plates in small, white, circular, shining, translucent colonies. It



lives in gall tissues but apparently its length of life in the galls has not been determined. Riker and Keitt (23) state that the organism has

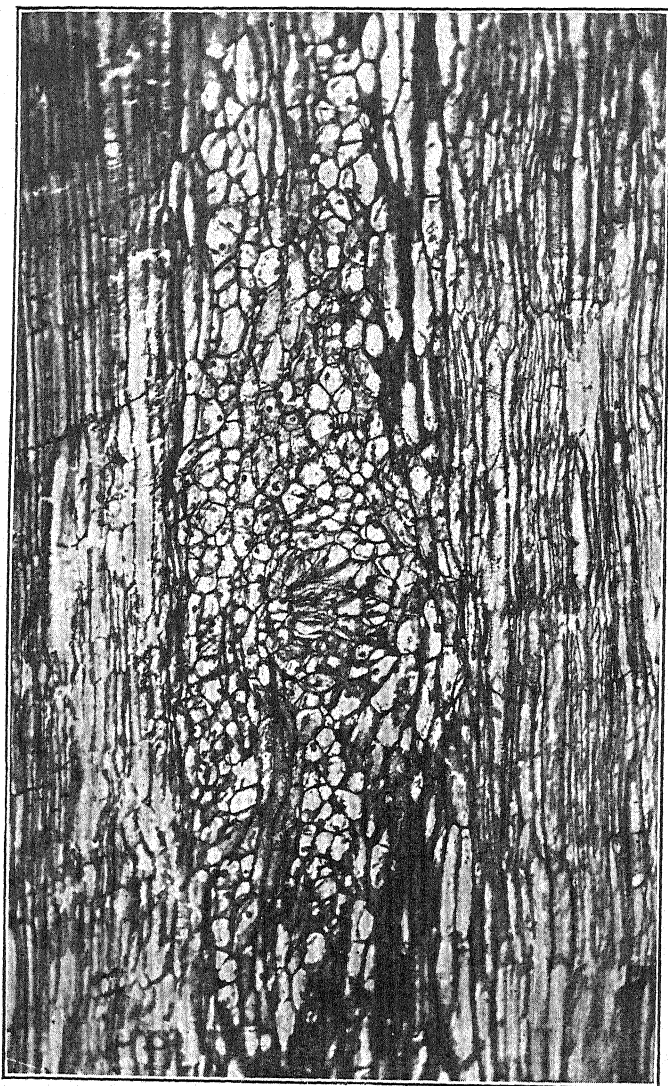


FIG. 25. — Longitudinal section of crown-gall "tumor strand" as found in Paris Daisy by Erwin F. Smith. (Published with the permission of the U. S. Dept. Agr.)

been isolated from overwintered galls. The organism is able to survive outside the host in the soil for an indefinite time, a few months (15) to

a year or more (16). It is disseminated by cultivation when liberated into the soil. The chief means by which it is scattered over long distances is in the transportation and transplanting of diseased nursery stock. Infection always occurs through wounds. Recent experi-

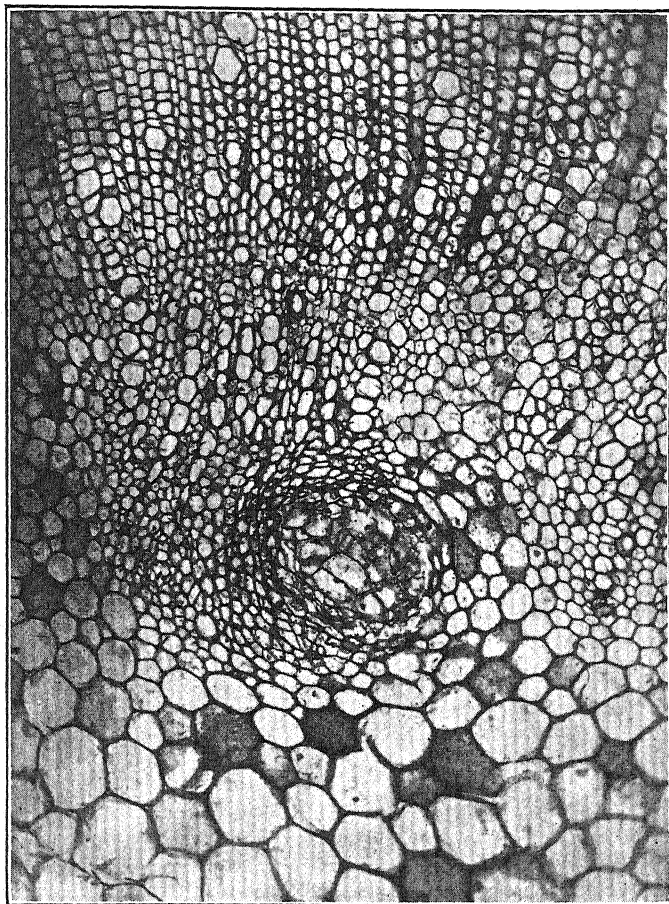


FIG. 26. — Cross section of Smith's tumor strand. (Courtesy of the U. S. Dept. Agr.)

ments (17) confirm the belief that infection cannot occur through the unbroken epidermis.

*Location of the bacteria in the host tissue.* — Until recently the opinion has prevailed that the organism lives entirely intracellularly in the host. Smith (31, 32) had great difficulty in demonstrating the bacteria in the host tissue at all, even after he had isolated it, grown it in culture,

and produced the disease again by inoculation. He finally succeeded in preparing sections which apparently showed the bacteria in small numbers inside the gall cells. More recently Riker (17, 18), by using a different technique, has shown that the crown-gall organism lives in great abundance in the intercellular spaces of invaded tissues (Fig. 27). Robinson's (24) work agrees with Riker in this respect.

**Pathological anatomy.** — The presence of the bacteria in meristematic tissues stimulates the cells to rapid division. The new cells may be small and spindle-shaped. Sometimes giant cells occur. The

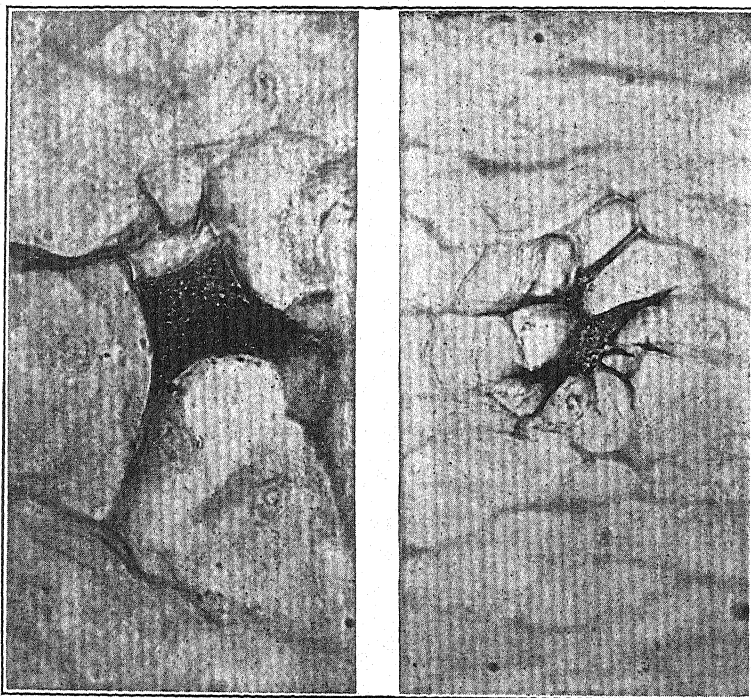


FIG. 27. — Photomicrographs of crown-gall bacteria in the intercellular spaces. Left, in a 10-day-old gall; right, in a 14-day-old gall. (After Riker.)

rapid division and multiplication of cells leads to overgrowth or hyperplasia. A section made through the plant so as to cut healthy and galled tissue shows that the galled tissue is easily distinguished from healthy tissue in its cell structure (32). With the growth of the tumor cells new vascular elements are formed, though these may be scanty and scattered promiscuously throughout the gall. If the tip of a young growing shoot is inoculated, tumors may appear on the elongating shoot

at points above the primary tumor which of course appears at the point of inoculation. Longitudinal and cross sections made through these growing tips (Figs. 25, 26) several days after inoculation show strands of differentiated cells extending vertically in the stem above the primary tumor and connecting it with the secondary tumor above in case other tumors form. Smith (32) called these structures "tumor strands" and thought they actually grew out from the original tumor, pushing their way up along the line of least resistance, and finally gave rise to the secondary tumors in much the same manner that human cancer behaves (27, 28). Doubt has recently been cast upon the tumor strand theory by Riker (17, 18), who states that the bacteria enter the liquid-filled intercellular spaces surrounding a wound instead of entering the cells themselves and that their presence stimulates the cells with which they come in contact to division, thus forming a gall. If they migrate for a considerable distance in a longitudinal line along these spaces the cells surrounding them divide in such manner as to form a region of differentiated cells (Fig. 28) such as Smith has designated a tumor strand. The formation of strands occurs only when infection takes place near the growing tip where stem elongation subsequently occurs.

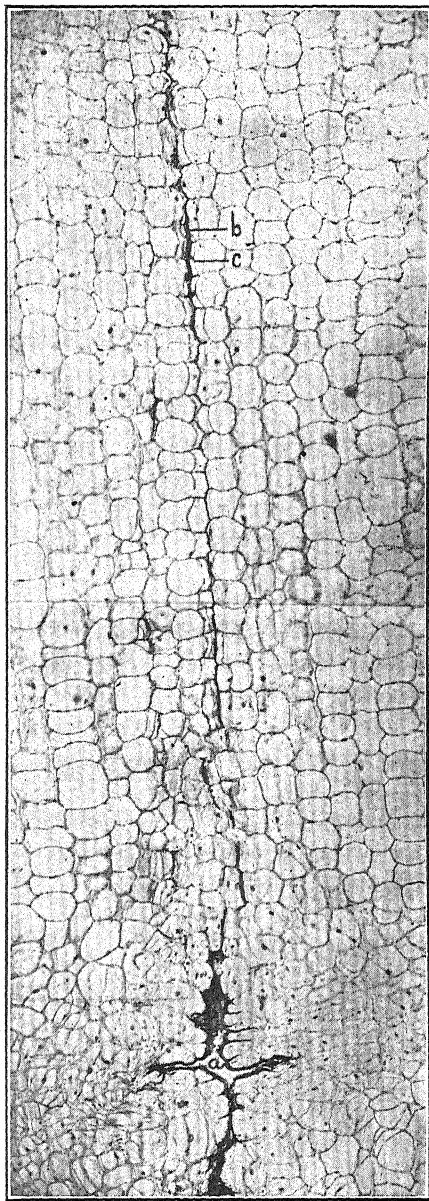


FIG. 28. — Longitudinal section of a tomato stem showing results of inoculation in the pith region. The needle puncture was made at *a*. The bacteria have migrated along the intercellular space, *b*. A few hyperplastic cells, *c*, may be observed about this invaded space. (After Riker.)

The formation of the "strand," then, is due both to the migration of the crown-gall organism in the liquid of the intercellular spaces surrounding the wound and to the elongation of the stem back of the growing apex. The work of Robinson and Walkden (24) in England and of Levine (10) in the United States corroborates Riker's views on this point.

**Control.** — The measures for controlling crown-gall must necessarily be directed largely toward the nursery since most crown-gall originates there. Distinction must also be made between the true crown-gall and the wound overgrowths which have been confused with crown-gall in the past. It has been found that the causal organism of crown-gall can enter only through wounds. Apparently any kind of wound may serve as an infection court, but wounds made by grafting and cultivation are probably the most common points of infection. Considerable work has been done on various methods of disinfecting nursery stock for the control of this disease. Melhus and Maney (11) in Iowa used bordeaux mixture, 8-8-50, and reported a reduction of 66 per cent in the amount of crown-gall. Preliminary experiments in Tennessee (25) indicate that sulfur applied to the soil just before the grafted trees are set in the nursery row may have a slightly beneficial result in reducing the percentage of crown-gall infections. Waite and Siegler (36) also report that crown-gall in grafted apple nursery stock can be controlled by disinfectants. It appears now that these workers may have failed to distinguish carefully between true crown-gall and graft-wound overgrowths, and that this may invalidate their findings to a great extent. Riker and Keitt (23) used a number of disinfectants but failed to prevent infection of tomato plants by the crown-gall organism.

Thus it would seem that the satisfactory prevention of crown-gall infections in the nursery is by no means an accomplished fact. In starting an orchard the grower must still insist upon nursery stock free from crown-gall. In the past, rigid inspection of nursery stock before it is shipped has been demanded. However, on account of the discovery that many gall-like and hairy-root-like growths are not due to a pathogene, and because of the fact that the crown-gall organism is thoroughly distributed throughout the country on a wide range of hosts, there is doubt as to just how rigidly the inspection and rejection regulations can be enforced without too great hardship upon the nurserymen or subjecting the orchardist to undue hazard (33). The recent work of Riker and Keitt should enable inspectors to discriminate more accurately between the infectious and the non-infectious types of galls or gall-like growths. In any case the grower who is starting an orchard should insist upon having the cleanest nursery stock available.

*Prevention of wound overgrowths.* — Riker and Keitt (23) have shown that many so-called galls occurring on apple nursery stock are of non-parasitic origin and are largely due to poorly fitted grafts (Fig. 24). They suggest that such malformations may be greatly reduced by (a) making properly fitted grafts, (b) wrapping the grafts securely with some suitable material such as waxed cloth or string, or adhesive tape, (c) callusing under the best conditions, and (d) being careful to avoid wounding in cultivation.

#### LABORATORY STUDY OF CROWN-GALL

**A. Symptoms.** — Examine specimens showing the different types of galls, including the typical underground galls, the hairy-root forms, and also aerial galls such as occur on grape vines and the canes of bramble fruits. Compare true crown-gall symptoms with various wound overgrowths, especially those occasioned by graft wounds, which are frequently confused with true crown-gall. Also compare with stem tumors on apple trees which were formerly thought to be crown-gall. (See reference 1.) Make drawings illustrating the different types of galls observed.

**B. Morphology and life history of the causal organism.** — Examine pure cultures of the organism and note its cultural characteristics. If stained mounts are available, examine with the microscope and note the rod-shaped body and the polar flagella. **Draw.** Read the text or other source of information and note the manner of *perpetuation, dissemination, and infection*. How long can the organism survive within the host? How long in the soil? How is it scattered from plant to plant? From one locality to another? Where and under what circumstances does infection occur?

1. **Cultures.** — If fresh, soft galls are available, try isolating the organism by the method described for bacteria in Chapter IV. Also inoculate susceptible potted plants, such as tomato, tobacco, castor oil plant, or daisy, and watch the development of the galls (see Chapter IV).

**C. Notes.** — Write complete notes on crown-gall, following the outline on page 152 in so far as it applies to bacterial diseases.

#### REVIEW QUESTIONS

1. Discuss Smith's theory of the nature and function of "tumor strands" in crown gall. (See text and references 31 and 32.)
2. Compare Riker's explanation of the origin of these so-called tumor strands with Smith's theory. (See text and references 17 and 18.)
3. Between crown-gall and what human disease has Smith suggested a possible relationship? (References 27 and 32.)
4. Are all knots found on the roots of plants crown-gall? Discuss other types of enlargements confused with crown-gall.
5. What is the nature of the injury caused by true crown-gall? (References 6 and 34.)
6. How much do galls on plants interfere with the water flow? (Reference 12.)
7. How long can the crown-gall organism survive in the soil independent of host plants? (Reference 15.)



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### Bacterial Blight of Beans

Caused by *Bacterium phaseoli* E F S.

This is one of the most widespread and injurious diseases of the bean crop occurring in the United States, ranking well up with anthracnose, a disease with which it is frequently confused. A third disease, *Rhizoctonia* rot, is also sometimes confused with the blight. Many growers do not distinguish between the various troubles which cause spots of one sort or another on the pods and leaves. Some names that have been applied indiscriminately to these different manifestations of disease are, pod spots, speck, blight, rust, scald and bean blight. The latter is the name most commonly applied to this particular disease.

**History and distribution.** — Apparently this disease was first known in America where it was mentioned by Beach (5) in New York in 1892. It was reported from New Jersey (12) in the same year with the further statement that it had been known in that state since 1886. It has since been reported from other countries but its origin is not definitely known. Since the first accounts of this disease came from America we shall have



to surmise that it is indigenous to this country until more certain knowledge is at hand. The causal organism was isolated, described and named by E. F. Smith (19) in 1897. Following this, brief accounts of the disease appeared by Halstead, Whetzel (21) and others. Still later more detailed studies on the life history and methods of control were undertaken by Edgerton (8), Muncie (16, 17) and Rapp (18). Burkholder (7) in 1924, published on varietal susceptibility of beans to blight.

Bacterial blight of beans is generally distributed throughout the United States, having been reported at one time or another from over three-fourths of the states in the Union, from New Hampshire to California and from Washington to Florida. It occurs in parts of Canada and probably in South America. In 1919 it was reported from both South Africa and the Philippines. A trouble which seems to have been the true bean blight was reported from France in 1899.

**Hosts and varietal susceptibility.** — The bean blight organism is confined in its attacks to species of beans, with few exceptions (9). There are other closely related forms of bacteria, possibly biologic races of the same species, which cause similar diseases on other genera of legumes closely related to the beans (13). As is the case with most other diseases all varieties of beans are not equally susceptible to this pathogene. A few observations on varietal differences had been made prior to 1919. In that year Burkholder (7) began a more extensive investigation into the possibilities of securing blight resistant varieties of beans. The results of three years experiments indicated varietal differences but no variety was found which was markedly resistant. Several of the less susceptible varieties were hybridized and at the time of publication, 1924, the work was being continued with the hope of ultimate success.

**Symptoms.** — The symptoms of the disease appear on any of the aerial parts of the plant (Fig. 29). The organism is even said to penetrate through the vascular system into the roots, but no surface lesions have been observed underground (6).

*On leaves.* — Infection is first indicated by minute specks on the underside of the leaf. These spots are at first dark green in color. As they enlarge, small dark green and water-soaked areas appear on the upper surface of the leaf. Many or few spots may appear on a single leaf, depending upon the severity of infection. As the spots enlarge the tissue turns brown and dries up. Sometimes the dead tissue becomes somewhat like parchment and more or less transparent (Fig. 30). The enlarging spots may coalesce and sometimes involve the entire leaflet. Finally the leaf withers and dies. It may cling to the plant for a time or soon become broken and ragged in the wind. If young

leaves are infected they may be distorted. Droplets of bacterial exudate may show on the surface. When this dries it forms a thin white or yellow incrustation on the leaf surface. Sometimes under hot,

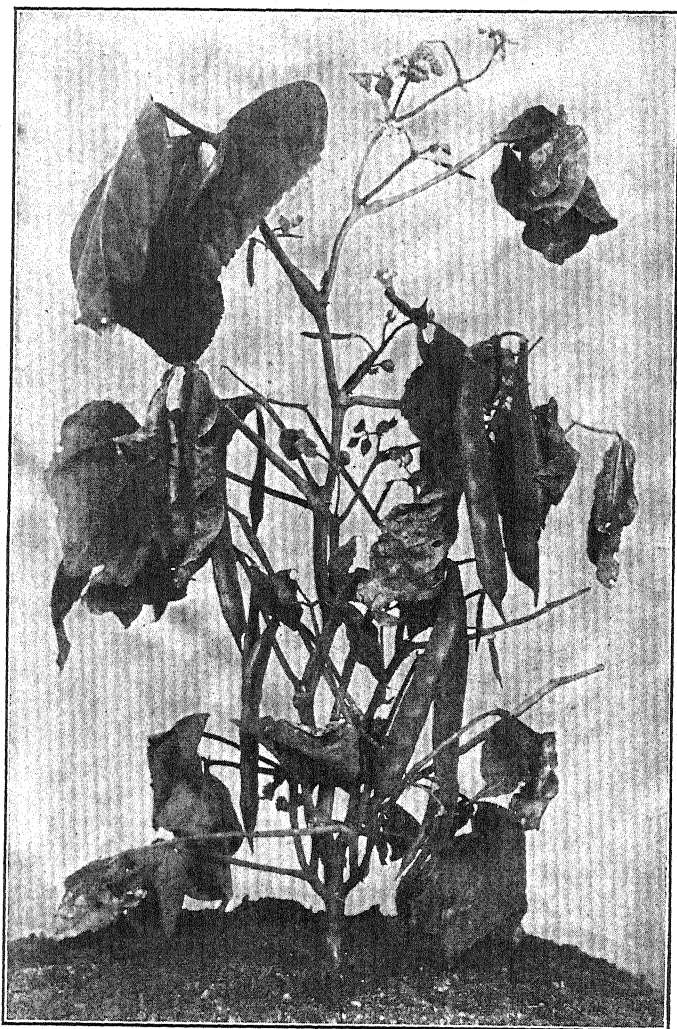


FIG. 29. — Bean plant affected with bacterial blight. (Photograph by Barss, Ore. Agr. Exp. Sta.)

humid conditions the leaves wilt down and blacken suddenly, appearing as if drenched with hot grease.

*On pods.* — The lesions on the pods start in much the same manner

as those on the leaves. Minute, dark-green, water-soaked spots appear. Drops of exudate usually form on these water-soaked areas. After a few days the spots turn reddish, then brown in color and sometimes

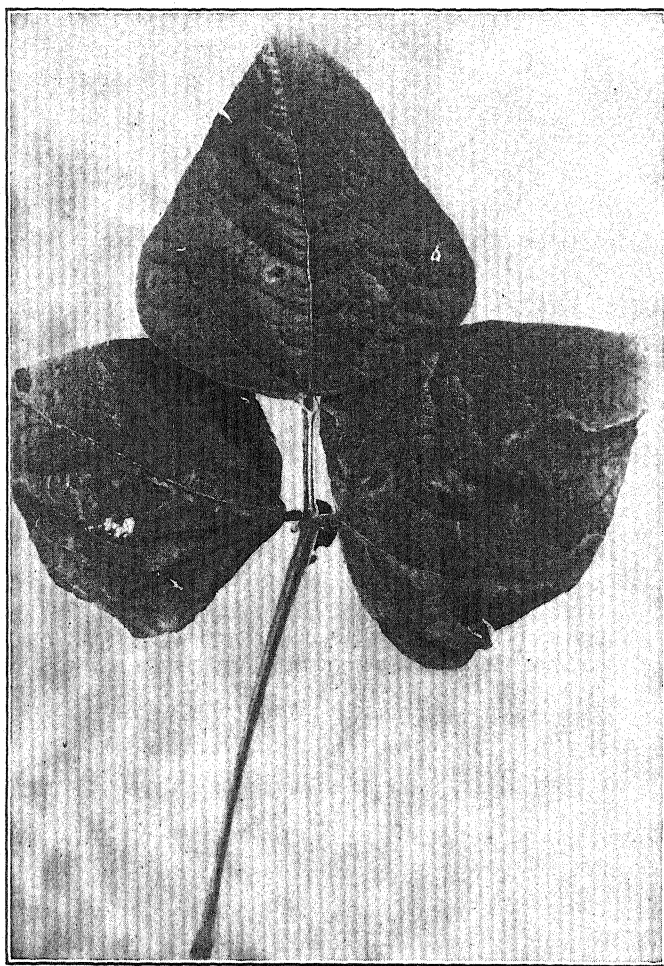


FIG. 30. — A bean leaf showing blighted areas.

somewhat purplish. Finally the surface of the spot becomes more or less sunken and irregular (Fig. 31). The spots vary in size, sometimes enlarging or coalescing until they cover the pod. The bacteria are able to invade the pod at any stage in its development.

*On seed.* — The seed is invaded either by the bacteria which penetrate directly through the ovary wall where the lesions occur or by organisms

which migrate in the vascular system of the plant and enter the seed through the hilum. Badly diseased seed may be much shriveled and discolored but less severely injured seed show only varying degrees of yellowish discoloration. Seeds which are so slightly yellowed as to be difficult of detection may carry the organism in abundance. Sometimes there is only a slight yellowing at the hilum. The cotyledons of germinating seed frequently show distinct blight discolorations when they emerge from the ground. This means that the seed harbors hibernating

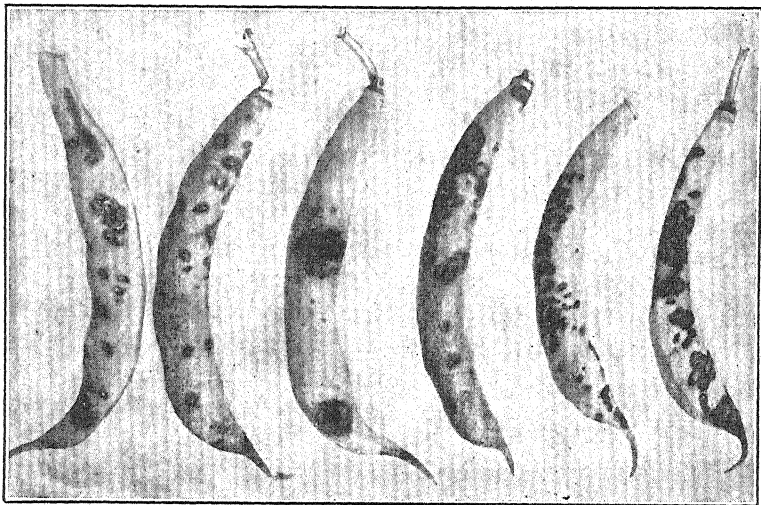


FIG. 31. — Bean pods showing symptoms of bacterial blight. (Photograph by M. W. Gardner, Purdue Agr. Exp. Sta.)

bacteria and that as soon as the seed resumes growth the blight-producing organisms become active again. The symptoms on pods and seeds as well as on leaves should be distinguished from those of anthracnose with which they are frequently confused. (See page 475.)

*On stem.* — Several different types of symptoms occur on the stem. On seedlings small water-soaked spots, similar to those occurring on the pods, appear. A seedling wilt also is frequently found. However, in this connection, attention should be called to a new wilt disease of beans recently described (14) which has probably been confused with the true bean blight at times in the past. Later in the season reddish-brown streaks or blotches appear on the stem. More or less indefinite canker-like lesions may occur. One of the most striking stem symptoms that sometimes appears is the girdling and breaking over of the stem at the cotyledon node or sometimes at nodes above the cotyledons.

The bacteria in this case attack the stalk at the point of attachment of the cotyledons or of the higher leaf petioles, forming a red cankerous lesion which encircles the stem and so weakens it that it topples over easily in a strong wind. If the stem is whipped back and forth considerably it may break off completely.

**Economic importance.** — Bean blight is one of the most serious diseases of beans in the United States. In former years it has usually been ranked second to anthracnose in importance, but reports of the Plant Disease Survey during the last few years (1921-1924) have given this disease first rank in point of percentage of loss caused to the bean crop in this country. In 1921 the loss from bean blight in Michigan alone was estimated (1) at 991,000 bushels or 25 per cent of the crop in that state. The loss for the whole United States was 1,040,000 bushels or 9.8 per cent of the entire bean crop of the country. In 1922 the loss in New York was reported (2) at 59,000 bushels, in Michigan 587,000 bushels, and in the United States 647,000 bushels. In 1923 the loss in Michigan (3) was 768,000 bushels or 10 per cent of the crop, while in New York it was only 122,000 bushels, which was 6 per cent of the crop in that state. In 1924 the loss (4) in New York was 10 per cent of the crop and in Michigan 5 per cent. California ranks with Michigan and New York as one of the three greatest bean-producing states in the Union

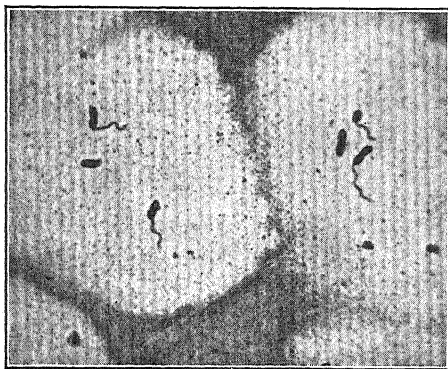


FIG. 32. — Flagellate rods of *Bacterium phaseoli*, the bean blight organism. (After Erwin F. Smith. Courtesy W. B. Saunders Co.)

but no comparative figures on the amount of loss from blight in that state are available for the years cited above.

**Life Cycle.** — This organism is a rod with rounded ends and is motile by one polar flagellum (Fig. 32). It is perpetuated in the seed and apparently also in diseased bean debris left in the field, and in the soil itself. There is abundant experimental as well as field evidence which indicates that

the organism hibernates in the seed. Muncie (17) and Rapp (18) both submit evidence that the organism also winters over on bean debris. The former also demonstrated that it overwinters in the soil. Rapp (18) is inclined to think that the organism cannot survive in seed for as much as two years, although he secured abundant evidence

of survival through one winter. Burkholder (6), on the other hand, states positively that two-year-old seed frequently harbors viable bacteria and in a few cases three-year-old seed produced diseased plants.

*Dissemination.* — It is evident, then, that the organism is disseminated in diseased seed. This accounts for widespread dissemination over long distances. In the field it is probably scattered from plant to plant by rain, insects and possibly on bits of dry pulverized leaves and dust blown about by the wind. The bacterial ooze which exudes from diseased leaves and pods affords an abundant supply of inoculum which can easily be carried from plant to plant by insects and by men, farm animals and implements passing through the field.

*Infection.* — Infection takes place locally wherever bacteria lodge on the surface of the plant. Entrance probably is effected largely through the stomata although slight wounds or punctures of any sort afford ideal infection courts. High temperature and humidity favor infection and spread. Periods of warm, wet, muggy weather are ideal for the inception of an epidemic. Apparently the incubation period is short. Smith (20) states that good results were obtained in artificial inoculations by leaving the plants in a moist chamber for 26- to 30-hour periods. Possibly heavy dews at night may be sufficient to bring about infection.

It has recently been discovered (6) that systemic infection as well as the ordinary local type of infection occurs in this disease. The bacteria in the diseased cotyledons or primary leaves of young seedlings sometimes invade the vascular system of the stem and there migrate to any part of the plant. Seedling wilt may result from this invasion. Sometimes the vascular infection is not severe enough to cause immediate wilting and death of the plant. Then the organism migrates to various parts of the plant. The effects are various. Dwarfing may occur. Lesions may appear on the surface at various spots or there may be no external lesions, for a time at least. Probably the most interesting thing about this type of infection is the fact that the organism may enter the seed through the vascular system without any external symptom showing on the pod. The only indication of seed invasion may be a slight yellowing of the hilum or even this slight symptom may be scarcely or not at all noticeable. This is a very important item from the standpoint of the selection of clean seed.

*Control.* — Completely satisfactory control measures for the bacterial blight of beans have not yet been worked out. Several different methods of approach to the problem suggest themselves and most of them have been the subject of much experimentation. These are: seed selection, seed disinfection, spraying, crop rotation, the use of aged seed, the use of resistant varieties, a special seed plot, and care in cultivation.

Two of these, seed disinfection and spraying, we may discard at once as having been proved of little or no value. The bean blight bacteria are not only on the surface of the seed but under the seed coats and between the cotyledons. Any treatment to be effective must be able to penetrate the seed and kill the bacteria without injuring the germinating power of the seed. No method of successfully surmounting this difficulty in the treatment of bean seed for blight has been devised. Even if the organisms were all on the surface, the soaking of bean seed in any kind of a poisonous steep has a tendency to make the seed coat slip and is therefore undesirable. Spraying the plants in the field to protect them from infection has also proved unsatisfactory. As a rule, bacterial diseases of plants are not amenable to control by spraying, and bean-blight is no exception to the rule.

Since the causal organism of blight hibernates and is disseminated chiefly in the seed it becomes necessary to get clean seed for planting. This can be done only by some method of selection since disinfection is of no avail. Several methods of selection suggest themselves: (a) the sorting out and discarding of diseased seed after harvest, (b) pod selection in the field, (c) securing seed from a disease-free field or locality, and (d) the growing of a special isolated seed plot by each bean grower. Sorting the seed after thrashing is impractical because the symptoms sometimes are so insignificant that all infested seed cannot be detected. Pod selection was formerly advised because it was thought if any disease existed sufficient to contaminate the seed it would be evident on the surface of the pod. Since the discovery of systemic infection, however, it is evident that many seeds may be invaded without any symptoms appearing on the pod. Therefore pod selection is unreliable. The only safe method of inspection is to examine the field carefully during the growing season to make sure there is no sign of blight anywhere in the field. Field inspection, then, is the only safe method of selecting bean seed free from blight. As a special aid in growing clean seed it is recommended that the grower have a special, isolated seed plot, as is also advised in combating potato diseases (see p. 92). Here a determined effort should be made to plant the cleanest seed available and by every possible means of clean and careful culture methods to keep the stock clean for use in seeding the commercial acreage each succeeding year.

Since the blight organism can hibernate on old bean straw and debris as well as in the soil, it is necessary to practice crop rotation in order to escape this source of infection. This applies to the special seed plot as well as to the commercial field.

The use of two- and three-year-old seed has been recommended. Certain investigators (18) claim that in their experiments such seed



gave a clean crop. On the other hand, there is evidence (6) that even three-year-old seed carries the organism in a certain percentage of the seed. Further investigation should determine this point satisfactorily.

Lastly, the question of blight resistance has been discussed in preceding pages. As yet no completely satisfactory blight resistant varieties are available but there is reason to hope that such will be developed in the future. This is always the ideal to which we should look forward in combating plant diseases.

Briefly summarized the following are the chief means of control at present available: (a) The use of clean seed secured by field selection, (b) crop rotation, (c) clean cultivation to exterminate possible weed hosts, (d) cultivation while plants are dry to avoid scattering the bacteria, (e) the use of a special seed plot, and (f) continued efforts to select and breed resistant varieties.

#### LABORATORY STUDY OF BEAN BLIGHT

**A. Symptoms.** — Examine leaves, stems, pods and seeds of beans affected with this disease. Observe partially blighted and totally blighted leaves. Describe the appearance of these leaves. **Draw.** If diseased stems are at hand note the lesions and make drawings to illustrate. On the pods note the small discolored spots and the more conspicuous older lesions. Are there any fruiting bodies of any kind evident? Compare these bacterial lesions on the pods with the cankers caused by bean anthracnose (see page 476). Note the discoloration of bean seeds from badly diseased pods. **Draw** diseased pods and seeds.

**B. Morphology and life history of the causal organism.** — If fresh, diseased plants are available, mounts of the bacteria may be made from the droplets of bacterial ooze frequently found on the surface of the pod lesions. Otherwise use pure cultures of the organism. Note the cultural characteristics; then observe the organism under the microscope. Is the organism a rod or some other form? How many flagella has it and where located?

**Life history.** — Find out where the organism overwinters, how it is disseminated in the field and from one locality to another, and how and under what conditions infection occurs. (See text.)

**Cultures.** — If specimens of diseased plants bearing live bacteria are available, try isolating and culturing the organism. Directions for carrying on this work may be found in Chapter IV.

**C. Notes.** — Write a complete account of bean blight following the general plan of the outline on page 152 in so far as it applies to this disease.

#### REVIEW QUESTIONS

1. What other bean disease is most easily confused with the bacterial blight? How may the two be distinguished? (See references 16, 17 and 21.)
2. Compare the bacterial pustule and the bacterial wilt of beans with the blight. (References 13 and 14.)
3. Discuss the transmission of the blight organism in seed, especially in connection with systemic infection. (See the text and reference 6.)



4. Why is not pod selection of bean seed a satisfactory method of securing seed free from blight bacteria?
5. Mention all the possible ways in which the organism may overwinter.

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### Bacterial Wilt of Cucurbits

Caused by *Bacillus tracheiphilus* E F S.

Of the four general types of bacterial diseases of plants this cucurbit disease is representative of the vascular wilt type. It is one of the more important maladies of certain members of the cucurbit family (Cucurbitaceae) and gives considerable trouble throughout many sections of the United States where cucumbers and muskmelons are extensively grown. The trouble was first noticed in 1893 near Washington and briefly described by Smith (10) who later (11), in 1895, described and named the causal organism. At that time very few bacterial diseases of plants were well known. More recently some intensive researches have been conducted to determine the methods of overwintering and dissemination. Rand (6) published the results of some investigations on dissemination in 1915. Rand and Enlows continued the work on transmission and control, publishing in 1916 (8) and in 1920 (9). Doolittle also has contributed to our knowledge of the overwintering of the organism (4) and of the susceptibility of varieties of cucurbits to wilt (5).

**Distribution.** — In 1920 the disease was reported to be present in 31 states of the Union and in 1925 the number of states in which the disease was said to occur had increased to 37. Its range as reported is from Vermont to Florida and westward to Minnesota and Texas. It may possibly occur also in California. It is said to be most prevalent in the latitude of Kansas, Kentucky, Indiana, Pennsylvania and Long Island, decreasing both northward and southward from this belt. In 1923 The Plant Disease Reporter (1) listed the disease as occurring on cucumber in many eastern, central and southern states, and on cantaloupe in 9 states including New York, Connecticut, Illinois, Iowa, Pennsylvania, Maryland, Ohio, Michigan and Wisconsin. In 1924 the same authority (2) reported bacterial wilt on cantaloupes in 10 states and on cucumbers in 16 different states. Outside of the United States it is said to occur in Canada, Japan, Transvaal, Russia and Germany.

**Hosts and varietal susceptibility.** — The disease is confined to the cucurbit family. It is known to attack cucumbers, muskmelons, pumpkins, squashes, gherkins and possibly watermelons, the latter very slightly if at all. It has been found also on certain wild forms of this family belonging to the genera *Cucumis*, *Cucurbita* and *Echinocystis*. Of the cultivated species and varieties the cucumber and muskmelon are the most susceptible and it is in these crops that the greatest losses occur. Squashes on the whole are much more resistant to wilt than cucumbers and cantaloupes and less loss from wilt is sustained by this

crop. Certain varieties, however, are highly susceptible. Apparently squashes are not very susceptible to strains of *B. tracheiphilus* from cucumbers and cantaloupes. To all intents and purposes the water-melons are immune. There is little evidence that natural infection occurs in this plant and artificial inoculations result in very slight wilting or none at all. Among the cucumbers there are no outstandingly resistant varieties although the American varieties are not nearly so susceptible as the European varieties (5). Among cantaloupes and squashes, however, there are varieties which show much more resistance than among cucumbers.

**Symptoms.** — The chief symptom of this disease is the striking and usually rapid wilting of the plant. The first indication of the disease usually appears as a flabby or wilted condition on a leaf which has been bitten by insects. The infection spreads outward from the infection



FIG. 33. — Cucumber wilt due to *Bacillus tracheiphilus*. (After Erwin F. Smith. Courtesy W. B. Saunders Co.)

court and then down the petiole into the main stem. Soon other leaves and finally the whole plant may wilt (Fig. 33). The wilted area eventually withers and turns brown. There is no rotting or other striking symptom connected with the disease on the intact plant. However, if the stem of a wilted plant is cut, a sticky fluid exudes from the cut ends of the vessels. This exudate is often quite milky and contains myriads of bacteria. A thin section under the microscope shows the spiral vessels filled with bacteria (Fig. 34). The presence of this viscid sap in the tracheae is an additional diagnostic character of the disease. The symptoms are not always as extreme on the squash or other less susceptible plants. In such cases infection sometimes results in a dwarfing rather than an immediate wilting of the plant.

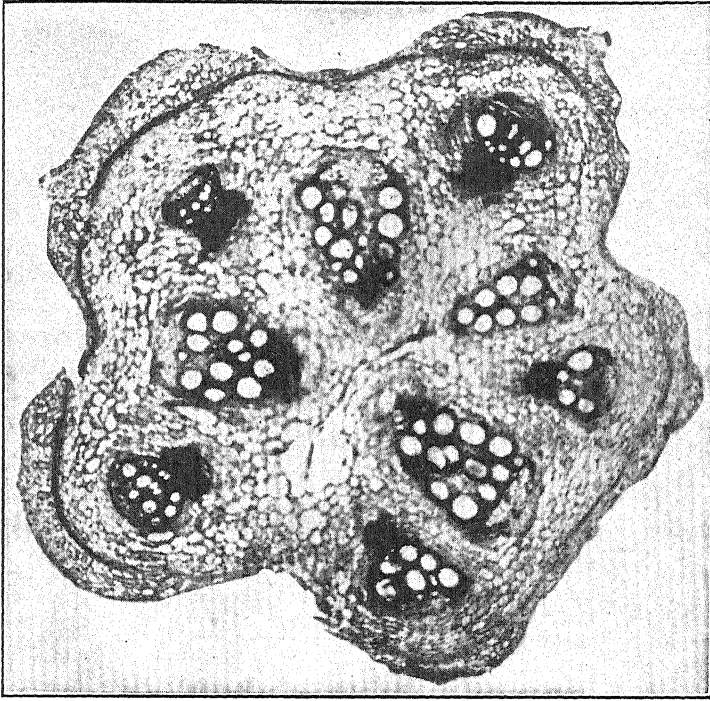


FIG. 34. — Cross section of a cucumber stem showing the spiral vessels in each bundle occupied by a mass of deeply stained bacteria. (After Erwin F. Smith. Courtesy W. B. Saunders Co.)

**Economic importance.** — It is not unusual for certain plantings of cucumbers to suffer an almost total loss from this disease. On the average, however, the loss is much less, but in the aggregate it amounts to a considerable percentage of the crop. In 1923 a loss of 25 per cent of the cucumber crop was reported (1) from Long Island. Illinois reported an average of 2.5 per cent loss with a maximum of 60 per cent in one field in the same year. In 1924 losses in the cucumber crop were reported (2) as follows: Illinois, 50 per cent in some fields with an average of 10 per cent; Ohio, 20 per cent; Iowa, 15 per cent; Pennsylvania, 5 per cent; and New York, 4 per cent. On cantaloupes the losses were: Iowa, 15 per cent; Ohio and Illinois, 5 per cent; and New York, 2 per cent. In 1925 the loss (3) on muskmelon in Ohio was 40 per cent in some fields; Iowa, 7 per cent; Indiana and New York, 3 per cent. On cucumbers the loss was: Kentucky, 20 per cent; West Virginia, 10 per cent; Iowa, 5 per cent; and in Ohio no definite loss was reported but the disease was present in 90 per cent of the plantings.

**Morphology and life history of causal organism.** — The germ causing this disease is a bacillus, therefore, rod-shaped and motile by means of peritrichiate flagella (Fig. 35). It is easily secured in culture from the viscid exudate from diseased stems. One of the most puzzling steps in the life history of the organism is its method of overwintering. All the

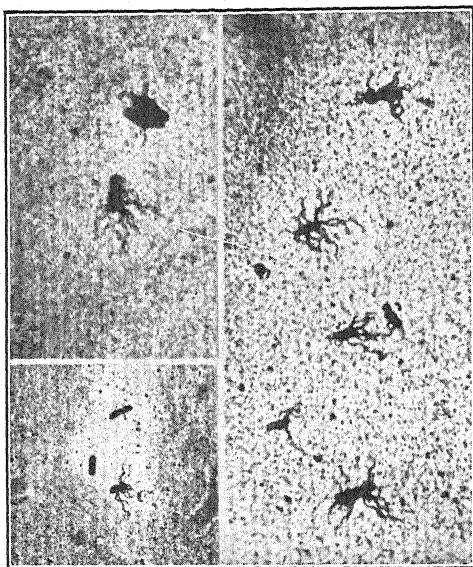


FIG. 35. — Flagellate rods of *Bacillus tracheiphilus*, the causal organism of cucurbit wilt. (After Erwin F. Smith. Courtesy W. B. Saunders Co.)

circumstantial evidence substantiates the theory that it hibernates in the cucumber beetles which infest these plants. It has been definitely proven that the striped and the 12-spotted cucumber beetles transmit the germs from one plant to another and the disease always seems to originate in the spring from the bites of beetles which have overwintered from the previous season. Attempts to hibernate the beetles artificially have been successful for a period of 6 weeks but in an experimental way no beetles have been brought through the entire winter

alive. Thus the evidence of hibernation of the organism in the beetles from one growing season until the next remains circumstantial. The organism has been isolated from the intestinal tract of beetles. Not all individuals of the two species of beetles carry the organism, however. Infection does not take place through stomata but only in wounds in the leaf. Artificial inoculations made in needle pricks result in infection. In nature the disease seems to originate only in the wounds made by the carrier-beetles. Apparently this is one of the few cases among plant diseases where a biological relationship exists between an insect and a plant-disease-producing organism. In such a relationship the organism passes a certain part of its life cycle in the insect host and the rest of the cycle in the plant host. (See Chapter XII.) In the greenhouse and wherever the vines are pruned, the pruning knife may be a means of dissemination. Smith (12) states that the disease appears to be worse

in warm, moist weather but that excessively hot weather retards its spread. Rand and Enlows (9) express the opinion that weather conditions do not have a marked direct influence upon the prevalence of the disease but that the presence of cucumber beetles bears a more direct relation to the occurrence of the malady.

**Control.** — Since the organism does not hibernate in the soil nor in seed, two usual methods of control, crop rotation and seed selection or disinfection, are eliminated at once as being of no avail in the control of this disease. Five possible avenues of attack remain: (a) spraying the plants with a fungicide, (b) elimination of the beetles, (c) roguing out diseased plants, (d) sterilization of pruning tools, and (e) the use of resistant varieties.

The use of bactericidal sprays for the control of bacterial diseases in general has never been very successful. There is some evidence that bordeaux mixture is fairly effective as a spray for cucurbit wilt. Where insects as carriers are concerned the problem is to either poison or repel them. Naturally the economical thing to do in this case is to develop a combination spray which will at the same time be effective against both the organism and its carrier. Since the cucumber beetles are chewing insects a poison is desirable. One of the best combinations yet devised for the control of cucurbit wilt is bordeaux mixture plus lead arsenate made up according to the 4-5-50-2 formula. Several dusts have been suggested for trial also. These dusts include one or more of the following poisons with a filler of gypsum or some form of lime: calcium arsenate, lead arsenate, paris-green, copper-lime-arsenate or black-leaf-40. All of these, of course, are primarily insecticides and are intended to control the disease by eliminating the insect carriers.

Since the beetles become contaminated by feeding upon diseased plants it follows that all wilted plants should be pulled out and disposed of in such manner that beetles cannot feed upon them again. Burning or burying is recommended. Where pruning of vines is practiced, as is especially the case in greenhouses, it is essential to take precautions against transmission of the germs by the pruning knife. The sticky fluid which oozes from cut stems is teeming with the bacteria and of course they are carried on the knife and inoculated into healthy plants which are subsequently pruned with the same knife. Pruning tools should be thoroughly disinfected before starting on each new plant. A solution of corrosive sublimate, 1-1000, has been recommended as a disinfectant. Incidentally cucurbits should not be grown in the vicinity of greenhouses used for growing cucumbers since the beetles are attracted to the plants grown outside and may thus find their way into the green-

house later. Insects should be carefully excluded from the greenhouse in any event.

The prospect of wilt control through the selection and development of resistant varieties does not seem very promising at present. Extensive tests (5, 8, 9) have failed to discover any variety of cucumbers or cantaloupes which are strikingly resistant. While American varieties of cucumbers have proven less susceptible than European varieties, none of the American varieties exhibit as much resistance as could be desired in stock which might be used in breeding up immune varieties. Among the squashes varieties of greater resistance are found but there does not seem to be the great need for resistant varieties here that exists with regard to the cucumbers.

#### REVIEW QUESTIONS

1. Which one of the four general types of bacterial diseases is illustrated by the cucurbit wilt?
2. What crops are known to be more or less susceptible to this disease?
3. What is the chief symptom of this malady? Upon cutting across a diseased stem what other striking sign of disease becomes evident?
4. Of what economic importance is the disease?
5. Describe the causal organism. How may it be secured in culture?
6. Discuss the overwintering of the organism. What is meant by "internal biological transmission" of disease organisms by insects?
7. What insect is suspected of transmitting this organism and what is the evidence against it?
8. Can the organism be transmitted in any other manner than by insects? If so, how?
9. Discuss the various items which should be taken into consideration in devising effective control measures.

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### Soft-rot of Carrot and Other Vegetables

Caused by *Bacillus carotovorus* L. R. Jones

Many fleshy vegetables are subject to soft-rots caused by bacteria. There are possibly several species of bacteria, or perhaps strains rather than distinct species, causing soft-rots of various vegetables and other herbaceous plants. These rot organisms attack especially those roots, fleshy stems, fruits and other parts of plants which consist largely of succulent parenchymatous tissue. The best known species of soft-rot bacteria is *Bacillus carotovorus* which causes the soft-rot of carrots but is by no means confined to this vegetable. It has been reported as causing a rot on more than two dozen other species of plants.

**History and distribution.** — *B. carotovorus* was first carefully described and named by Jones (6) in 1901. For at least ten years previous to this time bacterial soft-rot of various vegetables had attracted attention and had been studied and described but the specific organism had not been determined. During the next few years following Jones' work on this organism several other workers in Europe and America described either this species or closely related species or strains under various names. In 1902 van Hall in Holland described an organism which he isolated from iris bulbs under the name *Bacillus omnivorus*. In the same year Harrison (5) described *Bacillus oleracea* as causing a soft-rot in cauliflower and related plants. In 1904 Townsend (11) published a description of a soft-rot organism found in calla lily, which he named *Bacillus aroideae*. In 1910 Giddings described a soft-rot of muskmelon and named the organism *Bacillus melonis*. Still other species were named but need not be mentioned here. Investigations carried on by Harding and Morse (4) and by Massey (8) indicate that all of the forms of bacteria mentioned above can be referred to at most two species, namely, *B. carotovorus* and *B. aroideae*, and that possibly *B. aroideae* is only a strain of *B. carotovorus*. Thus the latter species is by far the most important of the soft-rot bacteria if not the only species concerned.



This disease is widespread in the United States, having been reported from a number of states. Either this species or closely related species occur in Europe as indicated by the publications on similar diseases in various countries of Europe. No doubt this or kindred species exist all over the world.

**Hosts.** — The host on which Jones made his chief studies was the carrot but the disease is by no means confined to this species of plant.

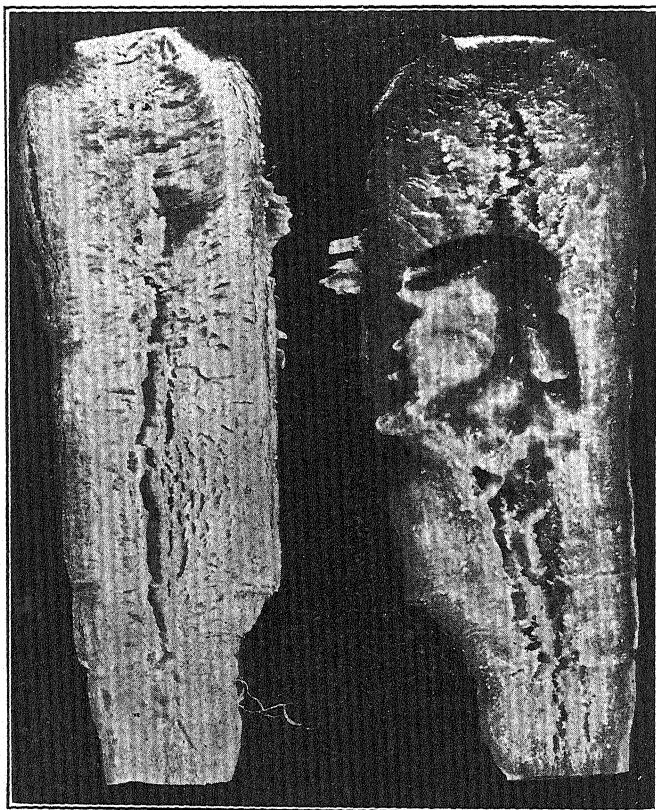


FIG. 36. — Soft-rot of carrot caused by *Bacillus carotovorus*. Artificial inoculation. (After Erwin F. Smith. Courtesy W. B. Saunders Co.)

Jones himself proved by inoculation experiments that the bacillus will invade a dozen other vegetables. If all the cases of soft-rot reported up to the present time prove to be due to *B. carotovorus* or strains of it the list of susceptible host plants will include at least the following: artichoke, asparagus, brussels-sprouts, cabbage, calla lily, carrot, cauliflower, celery, cucumber, egg-plant, horseradish, iris, kohlrabi, lettuce,

muskmelon, onion, parsnip, pepper, potato, radish, rhubarb, rutabaga, salsify, tomato and turnip.

**Symptoms.** — The first indication of infection by the soft-rot organism is the appearance of water-soaked translucent areas. This condition soon progresses into a soft slimy rot (Fig. 36). The color in various cases changes to a brown, a clay-color, or a dark color. In other cases there is no staining. The enzymes of the organism dissolve the middle lamellae thus permitting the cells of the tissues to separate (Fig. 37). This accounts for the softness, and the rapid and total disintegration of the invaded tissues. The epidermis in many cases remains intact but the decaying root shrivels and exudes a grayish sap filled with bacteria. The infection usually begins at the crown or root tip. In carrots, the core rots more rapidly than the outer part of the root. In hosts other than the carrot the general symptoms are much the same.

**Economic importance.** — No definite figures are available as to the amount of loss caused by this trouble but since so many different kinds of plants are susceptible the damage in the aggregate is considerable. Carrots suffer most when left in the ground after maturity or when stored under conditions not conducive to holding the organism in check. Celery sometimes suffers severely from the soft-rot. Other plants with fleshy roots as turnips and rutabagas are sometimes subject to extensive rotting from this source. Some complaint has been made of serious trouble in certain ornamentals, such as the Calla lily and the Iris, which are propagated from fleshy underground bulbs, rhizomes or other structures.

**Morphology and life history of the organism.** — The soft-rot organism, *Bacillus carotovorus*, is rod-shaped, with two to five peripheral flagella, and measures  $0.6-0.9 \times 1.5-5 \mu$ . Surface colonies on agar are gray-white with surface slightly elevated, smooth and shining. The organism is perpetuated in plant parts anywhere, in storage or in the field. It is disseminated largely in the handling of produce. If a rotted plant is smeared on the healthy ones with which it comes in contact the germs are carried wherever the produce is transported and further infection may appear at any time. The organism is liberated into the soil by decaying parts left in the field and may be scattered about by cultural operations. Infection occurs mostly in wounds and is favored by warm humid conditions. The optimum temperature for growth of the organism is  $85^{\circ}\text{F.}$ , but it can grow to some extent at lower temperatures.

**Pathological anatomy.** — The bacteria causing this trouble are able to secrete an enzyme which acts particularly in dissolving the middle lamella without breaking down the cellulose wall of the cell. This allows the cells to separate and thus the tissue disintegrates without the

individual cells losing their identity. The protoplast is evidently killed, however, since it is more or less collapsed within the cellulose shell of the cell wall. A section through decaying tissue at the right stage shows the cells separated and the spaces between filled with bacteria (Fig. 37). The organism is seldom if ever found within the cell lumen unless the wall has been mechanically ruptured.

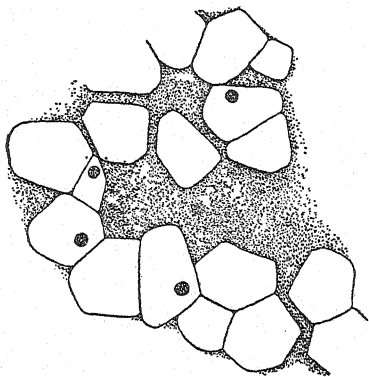


FIG. 37. — Section of turnip attacked by the soft-rot organism, showing the macerated tissue and the bacteria occupying the spaces between the separated cells.

**Control.** — The nature of this disease necessitates the application of certain measures of control. The practices which seem best suited to reduce to a minimum the losses from soft-rot are: (a) crop rotation, (b) care in handling, (c) exposing the crop to sunlight and drying before storing, and (d) cool, dry storage quarters or shipping carriers. If the soil in a field is believed to be infested with this organism, crops that do not suffer from this disease, such as cereals, legumes, or other forage crops

should be grown for a period of years. Since wounds are the chief port of entry for the bacillus, careful handling should be encouraged in order to avoid cutting and bruising as much as possible. It is known that sunlight and dry conditions are good germicides, therefore if root crops especially are allowed to dry in the sun after being dug the danger from rot is reduced. The minimum temperature at which this organism is active is about 39° F.; therefore, if storage quarters or cars are kept at a temperature below this point very little rot should develop.

#### LABORATORY STUDY

**A. Symptoms.** — Examine specimens of carrots, turnips or other vegetables affected by the bacterial soft-rot. Note the color, odor and general consistency of the affected parts, both in early and later stages of decay. Mount a bit of the decaying tissue from the border of the rotten area and examine with the microscope. Observe how the cells are separating. What causes the tissue to disintegrate in this manner?

**B. The organism.** — In the mount just made observe the myriads of bacteria in the spaces between the separated cells. If facilities and time permit try isolating and culturing the organism by the method described in Chapter IV. Try inoculating various vegetables either with the pure cultures or with material taken directly from the decaying specimens.

C. Notes. — Write notes describing the symptoms, life history and control of this disease.

#### REVIEW QUESTIONS

1. Name a list of vegetables which are subject to this disease.
2. Describe the symptoms of soft-rot.
3. How is the organism perpetuated and disseminated?
4. What conditions are necessary for infection to occur?
5. Describe the manner in which decay of plant tissues is effected by the organism.
6. In a mount of diseased tissue viewed under the microscope, where are the bacteria always found, unless the cell walls are ruptured?
7. Discuss control measures.

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### Bacterial Gummosis of Cherry

Caused by *Bacterium cerasi* Griffin

So far as known this is a disease of local distribution in the United States. It has been reported only from the Pacific coast region, namely in western Oregon and Washington, and in certain parts of California. In that part of Oregon and Washington lying west of the Cascade mountains it is a very serious disease of certain varieties of sweet cherries, assuming the proportions of a limiting factor in the production

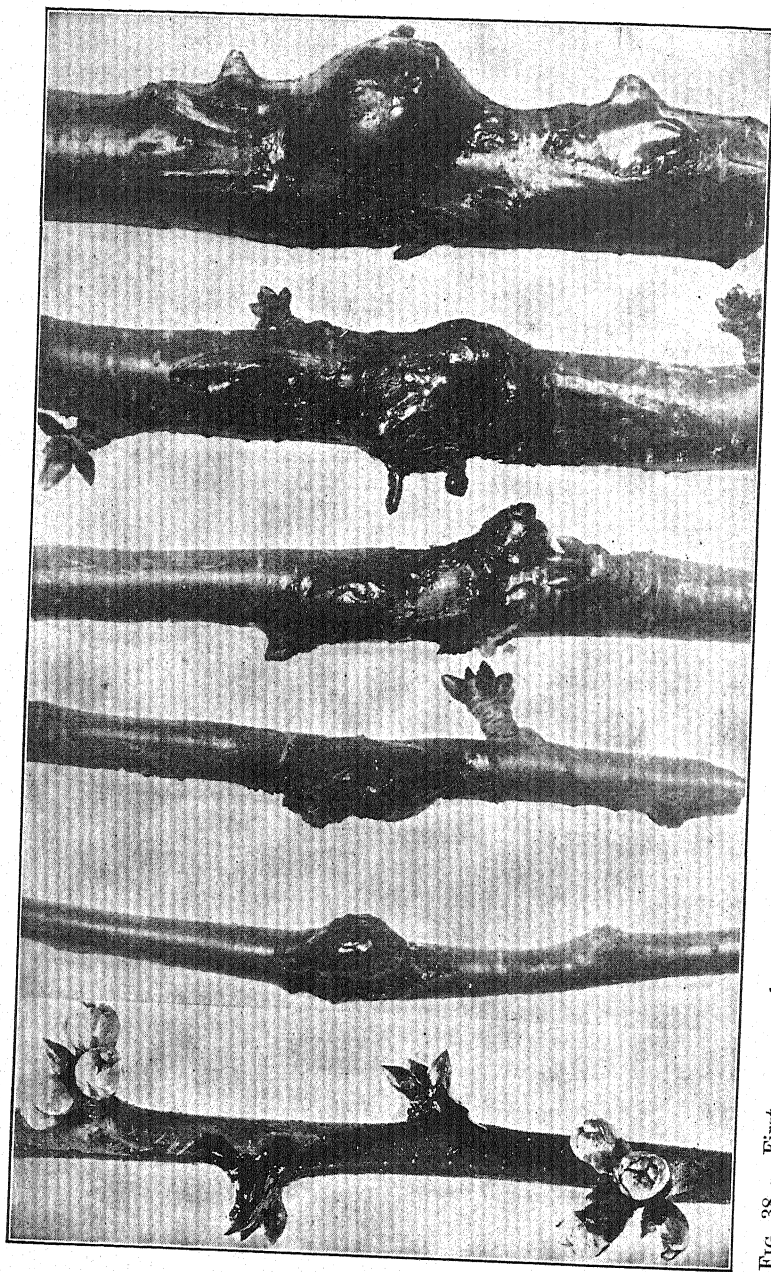


FIG. 38. — First season cankers on cherry, caused by *Bacterium cerasi*, showing excessive exudation of gum. (After Barss.)

of this fruit. The disease is also sometimes found on apricots and other stone fruits. The symptoms consist of large cankers (Figs. 38, 39), which often girdle branches or even the trunks of younger trees. The buds are also frequently blighted (Fig. 40). Copious exudation of gum usually occurs on the diseased areas giving rise to the term "gummosis" which is usually applied to this disease. The causal organism is supposed to be disseminated by insects and by rain. Infection apparently occurs in the fall and the cankers grow during the dormant season of the tree. When the tree resumes full activity in the spring the cankers cease to enlarge. The only practical means of control known is surgery and the use of resistant stocks for the trunk and scaffold branches on which the desirable varieties are top-worked.



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#### Citrus Canker

Caused by *Pseudomonas citri* Hasse

This disease is a native of the Orient. It is known in Japan, the Philippine Islands and parts of Eastern Asia. It was introduced into the Gulf region of the United States on nursery stock from Japan. It has caused consternation in the citrus fruit growing sections of this country, and strenuous efforts are being made to stamp it out.

FIG. 39. — Old open canker as it appears two or three years after the first invasion by the cherry bacteriosis organism. (Photograph by Barss, Ore. Agr. Exp. Sta.)



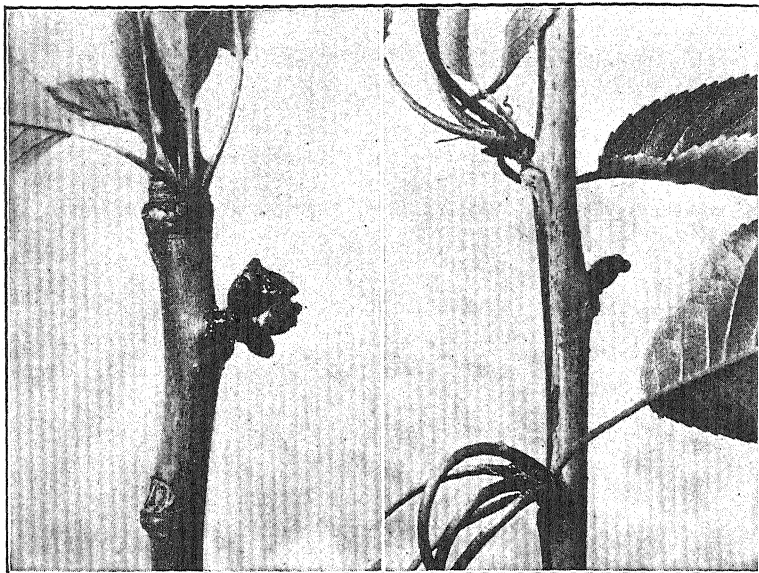


FIG. 40. — The blighting of buds and spurs resulting from attacks by the cherry bacterial gummosis disease. (Photograph by Barss, Ore. Agr. Exp. Sta.)

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### Bacterial-blight of Walnut

Caused by *Bacterium juglandis* (Pierce) E F S.

This disease is confined to the English or Persian walnut. It has long been known in California where this walnut is extensively grown. It occurs also in Oregon and has been reported from the eastern United States where the English walnut is grown in a limited way. The disease

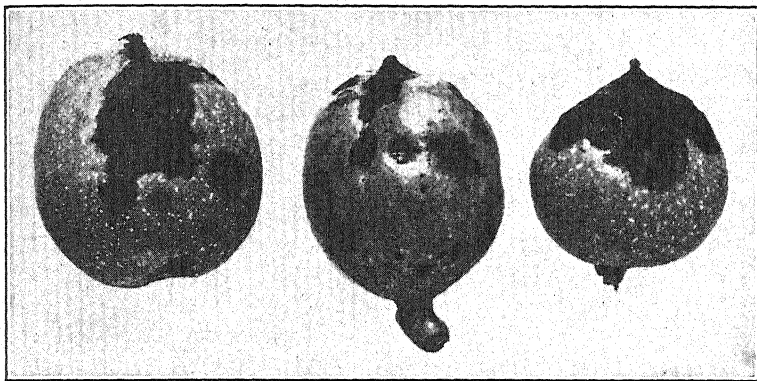


FIG. 41. — Bacterial blight of English walnuts. Symptoms on the half-grown nuts. (Photograph by Elmer and Barss, Ore. Agr. Exp. Sta.)

frequently causes great damage, especially when it attacks the fruits (Fig. 41). The damage to leaves and twigs is not usually serious.

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### Blackleg of Potatoes

Caused by *Bacillus atrosepticus* van Hall

This is a common disease in both Europe and America but it does not usually rank as one of the major potato diseases. In special cases considerable loss results from this trouble. Blackleg is characterized by a



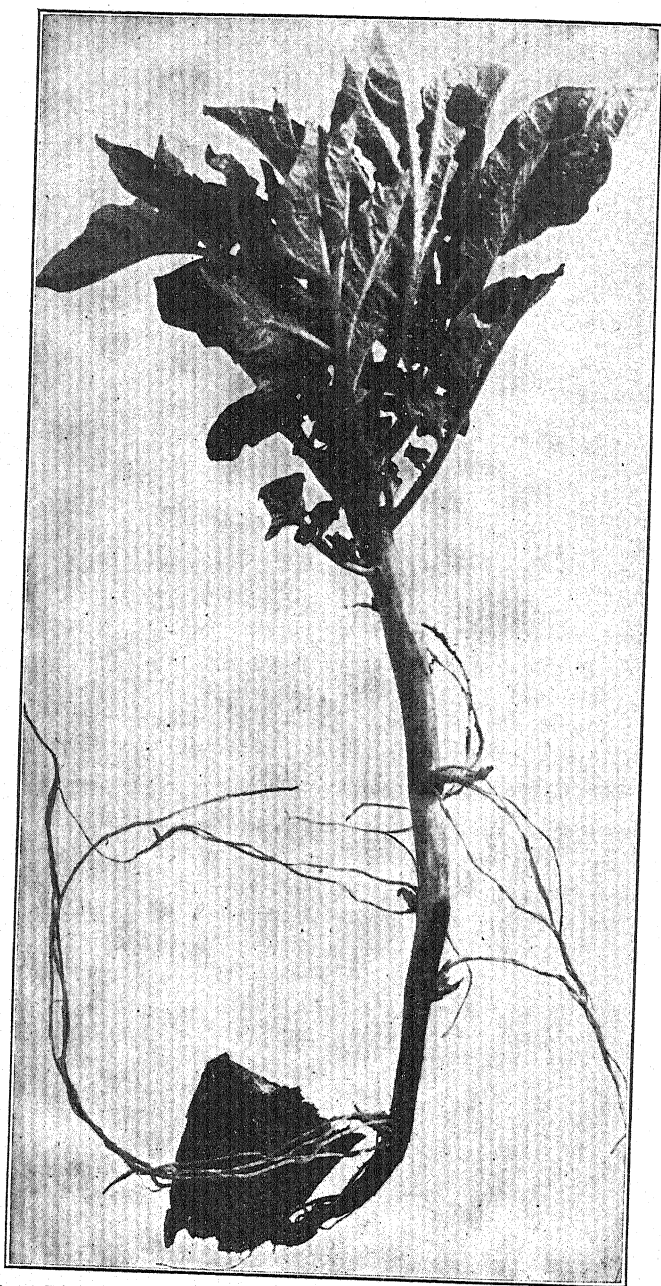


FIG. 42. — Blackleg of potato. (After McKay, Ore. Agr. Exp. Sta. Cir. 24.)

very black-rot at the base of the stem (Fig. 42). The necrosis affects mainly the parenchyma of both cortex and pith, finally rotting the stem off entirely in many cases. The tubers also are affected with a soft slimy rot. The black stem lesions suggest the common name of the disease.

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## Black-rot of Crucifers

Caused by *Bacterium campestre* (Pammel) E F S.

In some cabbage-growing sections of both America and Europe, this disease is more or less prevalent. It is largely due to invasion of the vascular system by the causal organism, which results in wilting of the plants and a blackening of the vascular bundles in both stem and leaf.

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## CHAPTER XVI

### DISEASES CAUSED BY FUNGI — PHYCOMYCETES

By far the greater number of parasitic diseases of plants are caused by fungi, that group of the Thallophytes which have no chlorophyll and consequently cannot carry on the work of photosynthesis. The fungi, therefore, are dependent plants and live either saprophytically or parasitically. Those fungi which live entirely upon dead organic matter are called *saprophytes* while those which live upon other living plants are known as *parasites*. Many of our most important plant diseases are caused by fungi which are strictly parasitic. On the other hand there are many diseases caused by fungi which are facultative saprophytes, that is, at one stage of their existence they are parasitic but have the ability to spend a part of their life cycle as saprophytes. Some fungi can live indefinitely on dead organic matter and carry on the reproductive processes, but may, under certain conditions, drop the saprophytic habit and become parasites. (See Chapter IV.)

One of the most striking differences between fungi and the two groups of parasites previously discussed (slime molds and bacteria) is found in the vegetative phase, which, in fungi, consists of branched fungous threads. These filaments are known as *hyphae* (singular, *hypha*), and, considered collectively, make up the *mycelium*. The mycelium grows either upon or within the substratum, and hyphae, or branches therefrom, penetrate either between the cells or into the cells and constitute the absorptive organs of the fungus. Reproduction in fungi normally takes place by means of spores produced either asexually or sexually, or both. There are certain exceptions to this rule, however, such as sclerotia and vegetative mycelium, and some of these will be considered in connection with the specific diseases discussed later. In the succeeding pages the fungi will be considered as composed of four classes, namely, Phycomycetes, Ascomycetes, Basidiomycetes, and Fungi Imperfecti. The distinguishing characteristics of each class, both vegetatively and reproductively, will be discussed under each class heading.

#### Diseases Caused by Phycomycetes

The Phycomycetes are characterized by their alga-like reproductive habits and their non-septate mycelium. Many serious plant diseases

are caused by fungi belonging to this class. Two well-known diseases caused by fungi belonging to one of the lower orders of the Phycomycetes are the potato wart disease (*Synchytrium endobioticum*) and the alfalfa crown-wart disease (*Urophlyctis alfalfae*). Probably the most important order of this class, from the plant pathological point of view, is the Peronosporales to which the downy-mildew fungi belong. The damping-off fungus, *Pythium*, may also be classed here or in a closely related order. In the following pages the damping-off disease and several of the downy-mildew diseases, namely, the late-blight of potato, downy-mildew of grape, and onion-blight or downy-mildew are discussed in some detail, and a list of references on some of the other diseases caused by fungi of this class is appended.

### Damping-off

Caused by *Pythium debaryanum* Hesse, and other fungi

Damping-off is a term applied to a very common trouble in which seedlings are attacked at the surface of the ground in such manner that the stem is partially or completely rotted and the plant suddenly topples over in a characteristic manner. There are several other species of fungi besides *Pythium debaryanum* which frequently cause damping-off. Some of the more important are species of *Fusarium*, *Corticium* (*Rhizoctonia*), *Sclerotinia* and *Botrytis*. Certain species of these genera will be discussed in other connections later in this book. In this exercise, however, the damping-off disease will be discussed largely in a general way regardless of the particular species of fungus causing the trouble, since many of the symptoms and the problems connected with this type of disease are similar whether the causal organism be *Pythium*, *Fusarium*, *Corticium* or any of the others.

**History and distribution.** — Damping-off diseases have been known for a long while both in Europe and America. In Europe, troubles probably due to some of the damping-off fungi have been known for over a hundred years. Near the middle of the nineteenth century a disease of this type due to the *Rhizoctonia* fungus was described in Germany. Hesse (14) described *Pythium debaryanum* in 1874. Hartig and others in Germany made extensive observations on diseases of this nature occurring on seedlings of various non-coniferous forest trees, including the maple, beech, ash and sycamore. Similar troubles were noted in England at an early date also. In 1883 Ward (21) published some observations on the genus *Pythium* which is one of the most

important genera of damping-off fungi. In the United States this type of disease began to attract attention at the end of the nineteenth century and the beginning of the twentieth century. At first the observations on these troubles in this country were confined largely to the attacks on truck crops and other cultivated crops. As early as 1901 to 1905, however, the damping-off disease began to receive attention as a serious trouble in forest nursery seed-beds. Since that time a large percentage of the publications on damping-off in the United States have dealt with the problems arising from the attacks of these fungi on forest tree seedlings. Jones (16), Gifford (5), Hartley (9, 10, 11, 12, 13) and Spaulding (20), among others, have contributed materially to our knowledge of these maladies as they occur in the forest tree nursery.

Fungi of various species capable of causing damping-off probably occur throughout the world. They have been reported from practically all civilized parts of the world wherever agriculture is practiced to any extent. Diseases of this type are especially apt to be encountered where seedling plants of almost any kind are grown in large numbers in a crowded condition. This is practically true in seed beds where plants are propagated to be transplanted later to the field. Greenhouses, cold-frames and nursery seed-beds are ideal places for outbreaks of damping-off if moisture and temperature conditions are favorable. Sometimes damping-off occurs under field conditions where there is no crowding, but it is much less likely to be a serious problem here than in the plantings of densely crowded seedlings.

**Plants attacked.**—Almost any kind of plant may be attacked by damping-off fungi while in the young, tender, succulent stage of development. Whether or not a species is subject to damping-off depends, in a large number of cases at least, more upon the conditions under which the seedlings are grown than upon the particular species of plant concerned. A list of species on which attacks of various damping-off fungi have been reported includes clover, cucumber, cotton, sunflower, tobacco, tomato, lettuce, celery, sugar-beet, egg-plant, cress, cabbage, corn, wheat, pine, hemlock, Douglas fir (*Pseudotsuga*), beech, maple, birch, ash, locust, apple and citrus fruits. This list is by no means complete but is indicative of the wide range of species which are susceptible to attack by these fungi.

**Symptoms.**—The common name usually applied to these troubles is suggestive of the normal typical symptoms usually encountered, but in addition to this normal type there are certain other symptoms that are sometimes recognized as variations of the damping-off trouble (13, p. 9). These modified symptoms are mentioned particularly in studies of damping-off in forest-tree seedlings. They may manifest themselves

as germination losses, as late damping-off, or as a "blighting" of the top of the plant. The most striking symptom of normal damping-off is the sudden toppling over of the young plant (Fig. 43). In such cases the fungus attacks the plant at or near the surface of the ground and in a very short time actually rots the stem to such an extent that the plant suddenly falls over. This occurs so quickly in many cases that the plants are found in a prostrate condition even before the top has had time to wilt to any extent. If such plants are pulled they break off

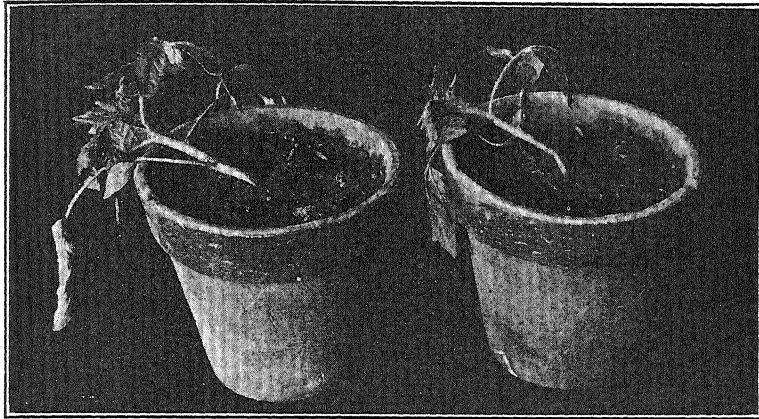


FIG. 43. — Young tomato plants showing the typical toppling-over symptoms of the damping-off disease. (Photograph by McKay, Ore. Agr. Exp. Sta.)

easily at the decayed point. After the plants have fallen over they may continue to decay, if the ground is wet and the air humid, until entirely rotted away, or if drier conditions prevail the top may wilt and dry up after toppling over. This later decay may be due to the further spread of the original organism through the tissues, or other fungi and bacteria may invade the sick plant. In some cases the radicle of germinating seeds is attacked as soon as it emerges from the seed coats so that the seedling never appears above ground. Heavy losses may result from this reduction in per cent of germination. In other cases attack is delayed until the stem has become woody and resistant to fungous invasion. The roots may remain susceptible longer than the stem and a root-rot may set in. Such seedlings do not fall over immediately but may be found dead while still standing. Not all seedlings attacked by the late root-rot type of damping-off succumb but a certain percentage of them survive. This type of trouble resembles drouth injury to some extent. The fourth type of injury consists of lesions appearing on the

cotyledons or upper part of the stem. These are due to invasion by the fungus before the seedling emerges from the soil and consist of killed areas of greater or less extent. When all the cotyledons or the upper part of the stem are involved, of course, this type of trouble is practically as fatal as if the stem were damped-off at the surface of the ground.

It will readily be recognized that the troubles caused by damping-off fungi comprise a large and important section of that larger group of diseases discussed under the general heading of "sick soil," in Chapter X. Not all organisms concerned in sick soil troubles cause a damping-off of seedlings but many of them do. Some of the fungi which cause serious damping-off in young seedlings also cause other important disease symptoms in the later life of the plant. *Rhizoctonia* is a good example of this (see under potato *Rhizoctonia*, p. 421). The fungi, *Gibberella saubinetii* and *Diplodia zeae* are other important organisms which cause serious seedling disease in wheat and corn and also are responsible for maladies appearing on these plants when they are more nearly mature and which cause enormous losses in the wheat and corn crops.

**Economic importance.** — The losses due to damping-off are confined largely to seed-beds, wherever they may be. In Europe the forest nurseries have been among the chief sufferers. Forestry is an old science in some sections of Europe, especially in Germany and France. In the nurseries where seedlings are grown on an extensive scale for reforestation purposes, outbreaks of damping-off have been recorded at different times for almost three-quarters of a century. Another type of damage caused by fungi of this nature is that due to attack of cuttings used in the vegetative propagation of certain plants. The Germans and French long ago complained of such troubles occurring on the cutting benches. In the United States during recent years since the government has begun to establish forest nurseries in connection with the national forest reserves, damping-off fungi have done considerable damage in the seed-beds. Among the truck crops and greenhouse crops the damping-off disease is frequently a serious trouble. No figures on losses are available but they are undoubtedly considerable.

**The causal organisms.** — A detailed account of the morphology and life history of all the fungi concerned in damping-off will not be given here. *Pythium debaryanum* will be described somewhat fully and some of the others mentioned more briefly. Some of the general phases of the life history will apply fairly well to any of the damping-off fungi listed above. *P. debaryanum* belongs to the class Phycomycetes, hence the mycelium is non-septate. The hyphae are much branched and are found both between and within the cells of the host tissue. Reproduc-



tion occurs both asexually by means of conidia, or zoöspores, and sexually by means of oöspores.

*Asexual reproduction.* — The asexual fruiting structures of *P. debaryanum* are formed either at the tips of undifferentiated branches of mycelium or as an intercalary cell at some point in a hypha (Fig. 44, *a, b*). The apex of the branch becomes swollen and a septum cuts off this enlarged tip. These enlarged, rounded cells readily break off from the

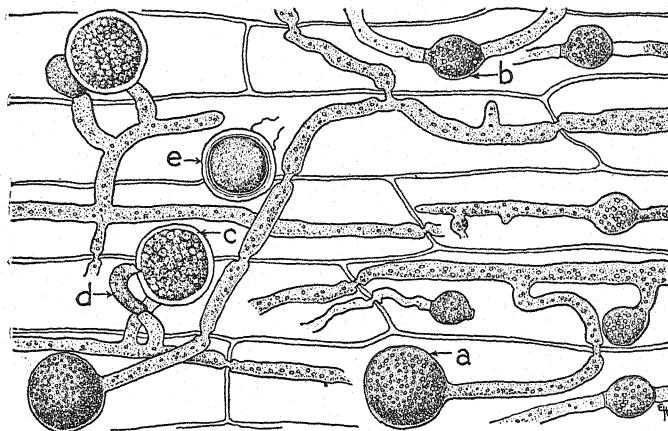


FIG. 44. — *Pythium debaryanum* growing in the tissues of an alfalfa seedling. (a) Terminal conidium; (b) intercalary conidium; (c) oogonium; (d) antheridium; (e) oospore.

parent hyphae and may function either directly as conidia or indirectly as zoösporangia. In the former case germination occurs simply by the outgrowth of a germ tube from the wall of the conidium. This germ tube may directly enter another host and establish a vegetative mycelium there. On the other hand these asexual reproductive bodies may germinate indirectly by the formation of several zoöspores from the protoplasmic content of each body. This process in *Pythium* is similar to the zoöspore formation in the downy-mildews which is described and illustrated in the following exercises. The zoöspores are bean-shaped masses of protoplasm with two lateral cilia. The zoöspore swims about for a time and finally, after coming to rest on the surface of a susceptible host, sends out a germ tube which penetrates the host in the same manner as those from the germinating conidia and thus causes infection.

*Sexual reproduction.* — This takes place in the same manner that it does in many of the algae and in a large part of the Phycomycetes among the fungi, namely, by means of the typical sex organs of these



forms, oögonia and antheridia (Fig. 44, *c, d*). These organs are cut off at the tips of hyphae by the formation of septa in much the same manner that the conidia or zoösporangia are cut off. The oögonium is a rounded body containing a denser central part, the oösphere or egg, and a less dense part surrounding the egg. The antheridium is also a somewhat enlarged terminal cell but much smaller than the oögonium. A fertilizing tube grows out from the antheridium and penetrates the wall of the oögonium. Through this tube the contents of the antheridium, including a nucleus and some cytoplasm, pass into the oögonium where the nucleus from the antheridium fuses with the nucleus of the egg thus effecting fertilization. The fertilized egg then develops into a thick-walled oöspore (Fig. 44, *e*). The conidia or zoösporangia measure 20 to 25  $\mu$  in diameter while the oöspores range from 15 to 18  $\mu$ .

*Fusarium* and *Botrytis* reproduce by means of conidia, while *Rhizoctonia* perpetuates itself mainly by means of sclerotia but produces basidiospores occasionally. (See under potato *Rhizoctonia*, p. 425.)

**Life cycle.** — While any of the damping-off fungi may produce one or more spore forms under the proper conditions, they apparently are not dependent upon the production of spores for perpetuation. Since they are "sick soil" organisms this means that they may exist indefinitely in the vegetative state in the soil. They apparently live largely as saprophytes on plant débris or humus in the soil, and become parasitic when the proper hosts and the proper growing conditions are present. Some of them can attack such a wide variety of plants, including weeds, that there is no certainty that they will ever die out entirely. This ability to perpetuate themselves indefinitely in the vegetative state eliminates the necessity of a spore form in the life cycle. Nevertheless, spores are formed and probably function at times in reproduction. Such fungi as *Fusarium* and *Botrytis*, especially, may sporulate profusely above ground on killed plants and it is undoubtedly true that these spores may serve as a means of perpetuation and dissemination of the fungi. Just what part the different spore forms play in the life cycle of *Pythium* seems not to have been definitely determined.

Dissemination of the damping-off fungi may occur in various ways. They are so widely distributed in nature that it is doubtful if many soils are entirely free from all organisms of this kind. As has been mentioned some of them are probably further disseminated by means of spores. A very common source of contamination in seed beds, where the trouble is most likely to occur, is in the soil or compost used in making up the beds. Sometimes these materials are secured from an infested source and of course the organisms are introduced along with the soil or compost.

The mere presence of such organisms in a soil does not necessarily make an outbreak of damping-off inevitable. Certain conditions must exist before there is much danger of infection. As a rule only young, tender, delicate plants are subject to damping-off. The temperature and moisture conditions play a very large part in determining the severity of infection. Abundant moisture and a fairly warm temperature are conducive to invasion of the seedlings. Overwatering, thick sowing in the seed-bed, lack of proper ventilation, and too much shade are conditions which favor damping-off. The high temperature and humidity, and overcrowding of plants which frequently obtain in green-houses are ideal conditions for an outbreak of damping-off provided the right species of fungi are present in the soil.

**Control.** — Since the various fungi concerned in damping-off troubles are soil-borne and since the temperature and moisture factors of the environment play such an important part in determining the severity of infection, control measures for damping-off resolve themselves largely into two methods of attack, namely, the proper handling of the soil and the manipulation of the other environmental factors in so far as this can be done.

**Soil treatment.** — In securing new soil for seed-beds of course every effort should be made to find soil free from damping-off fungi. Since this is not always possible and since in large beds it is not always feasible to change the soil, it has been necessary to resort to soil sterilization when the beds harbor these organisms in sufficient numbers to interfere with the growing of the seedlings. There are two general types of fungicides used for this purpose, namely, heat and chemicals. When heat is used it is generally applied either in the form of hot water or steam. (For details see Chapter VII, p. 81.) The use of chemicals requires less equipment than does the use of steam in sterilizing soil. Formaldehyde and sulfuric acid are the two most commonly recommended substances for soil sterilization. The latter is in common use in forest nurseries where conifer seedlings are grown. In using sulfuric acid in forest nursery seed-beds, three-sixteenths fluid ounce dissolved in 1 to 2 pints of water is allowed for each square foot of seed-bed if the soil is heavy; a little less is used if the soil is sandy. This acid treatment can be applied without danger immediately after the seed is sown. In using the formaldehyde treatment about one-half fluid ounce dissolved in water is used per square foot. Formaldehyde should be applied several days before seeding and the soil covered with paper, tarpaulin or other covering until ready to sow the seed. Some recommend airing the soil out thoroughly after disinfecting and before planting the seed. Copper sulfate and zinc chloride are other soil disinfectants sometimes used with

more or less success. The formaldehyde and steam treatments are most commonly used in greenhouses and in seed-beds other than in forest nurseries.

*Environmental factors.* — In the greenhouse the temperature and humidity can be fairly well controlled. A system of subirrigation should be substituted for overhead sprinkling whether under glass or outdoors. When the plants are kept wet above ground damping-off is favored. The idea is to keep the plants above ground dry and also keep the surface of the ground dry without permitting the plants to suffer from lack of water. In forest seed-beds a thin layer of sand is sometimes sprinkled over the surface of the soil or the nursery is located on a sandy plot. This aids in keeping the surface of the ground dry. An alkaline reaction in the soil seems to favor the development of some damping-off fungi.

#### LABORATORY STUDY OF DAMPING-OFF

**A. Symptoms.** — Examine seedlings of tomatoes, beans, celery, cabbage, spinach, lettuce, pine, or any other plant affected with the damping-off disease. How large are the diseased plants? Is this a disease essentially of very young plants or one which usually attacks larger plants several weeks old? Note the part of the plant attacked, whether stem or root, above or below ground, or at the surface of the ground. Is there any variation in these details? Is the injury a rotting or simply a wilting of the affected plant? How long does it take the plant to collapse after the first invasion by the fungus? Make *sketches* to illustrate the different symptoms observed.

**B. The fungus.** — As noted in the text various species of fungi may cause damping-off. The one chosen for study here is *Pythium*. This is a fungus which reproduces both asexually and sexually. For a study of the fungus, bits of diseased seedlings or artificial cultures may be used.

*Vegetative mycelium.* — Tease out a bit of the soft decayed tissue of a diseased seedling and make a mount for microscopic examination. Find the mycelium ramifying through the tissues. Look for septa in the hyphae. Do you find any? To which class of fungi does *Pythium* belong?

*Asexual stage.* — If suitable material is available examine the asexual fruiting stage under the microscope. Note the zoösporangia, either terminal or intercalary. Look up, in text and references, the manner of germination of the sporangia. **Draw.**

*Sexual stage.* — Look for oögonia and antheridia. Are there any mature oöspores present? Search for specimens showing the conjugation of antheridium and oögonium. **Draw** any stages found. From the text and references learn the life history of the fungus. Look up other damping-off fungi, such as *Fusarium* and *Rhizoctonia*, and compare them with *Pythium*.

**C. Experimental.** — If soil infested with damping-off fungi is available, sow seeds of some susceptible plant in pots or flats, keep under favorable conditions for the development of the fungi, and observe the results when the seedlings emerge. If infested soil is not obtainable clean soil may be inoculated with cultures of the fungus.

**D. Notes.** — Write full notes on the symptoms, cause and control of the damping-off disease in plants.

## REVIEW QUESTIONS

1. Describe the various symptoms of damping-off.
2. At what stage in the development of plants are they particularly susceptible to this trouble?
3. Describe the morphology and life history of *Pythium debaryanum*.
4. Name some other fungi which may cause damping-off.
5. What environmental conditions are conducive to an outbreak of damping-off?
6. Name several garden and truck crops which are subject to this trouble.
7. Discuss this disease as it affects the practice of forestry.
8. Discuss control of this trouble as related to (a) greenhouse crops, (b) field crops, and (c) the forest nursery.

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### Downy-mildew or Blight of Onion

Caused by *Peronospora schleideni* Unger

This disease is one of the typical downy-mildews and the causal organism is quite closely related to those causing downy-mildew diseases of spinach, alfalfa and clover, mustard, cucurbits, lettuce, corn and potatoes. It is a well known and quite widely distributed disease of the onion and one which is capable of causing serious damage under favorable conditions. The ordinary onion is the most commonly attacked but the potato or multiplier onion and shallots are also sometimes infected by this fungus. Several different names have been applied to the disease among which are onion mold, white blight, white blast, downy-mildew, and onion blight. The last named term is probably the one most often used to designate this disease.

**History and distribution.** — The earliest account of the onion mildew fungus was written in 1841 by Berkeley (2) who described the organism and named it *Botrytis destructor*. In 1847 Unger named the same fungus *Peronospora schleideni* (11). In 1863 De Bary (4) changed the name to *Peronospora schleideniana* which name has had a very wide use among plant pathologists. On the grounds of priority, however, the name *P. schleideni* should be used to designate the causal fungus. The pathological literature of the last quarter of the nineteenth century contains frequent mention of this disease on the continent of Europe. In 1887 Shipley (8) reported the presence of onion mildew in the Bermuda Islands. The first definite record of this disease in the United States was made by Trelease (10) who reported it from Wisconsin in 1883, although it probably had existed here previous to that time. Dudley (5) found the disease common in New York in 1888. Thaxter (9) reported it from Connecticut in 1889, and in 1890 Jones (6) stated that it occurred in Vermont. In 1926 Murphy and McKay (7) published the results of five years investigations on the occurrence of perennial mycelium of the mildew fungus in onion bulbs. At the present time

the disease is widely distributed, occurring at least in the British Isles, in continental Europe, in Bermuda and in North America. It probably occurs in other countries as well. In the United States the disease is known in most sections where onions are grown extensively.

**Economic importance.** — The downy-mildew of onions is not equally serious every season nor in all localities. Epidemics may occur at irregular intervals with intervening years when the disease is relatively

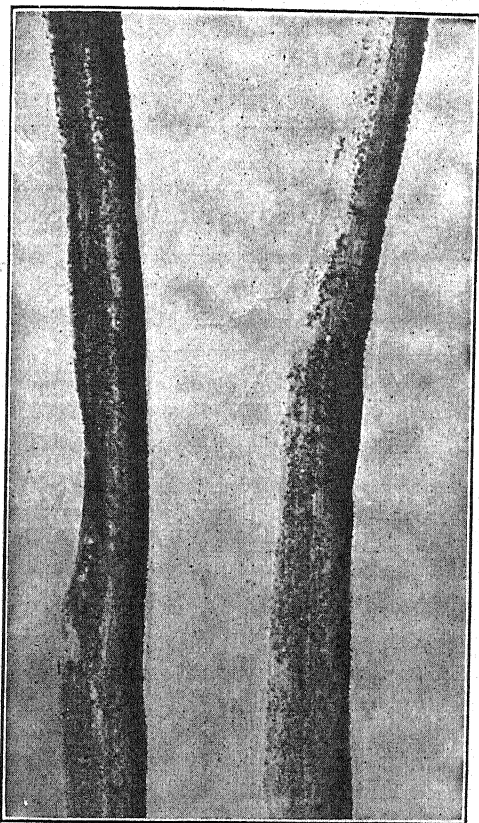


FIG. 45. — Onion leaves showing the conidial stage of the downy-mildew fungus. (Photograph by F. D. Bailey, Ore. Agr. Exp. Sta.)



FIG. 46. — Late stage of downy-mildew on onion showing the blighted leaves. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

unimportant or absent. Occasionally serious outbreaks occur when considerable damage is done. In severe cases the leaves are entirely blighted down (Fig. 46), and when this occurs before the bulbs are mature the loss may be great. In addition to the actual reduction in

yield due to the leaf injury caused by the mildew fungus, the blighted plants are rendered more susceptible to other fungi of the mold or rot-producing type and thus secondary invasions may occur causing further loss.

**Symptoms.** — The first signs of the disease appear before there is any striking manifestation of blighting on the leaves. These signs consist of the fruiting structures of the fungus which show as a violet or purplish colored coating of a downy nature over the surface of the leaves (Fig. 45). This may be described as a fuzzy or moldy growth and is most evident while the plants are wet with dew or rain. The occurrence of this fungous growth on the surface of the leaves may be observed usually while the leaves are still green and otherwise healthy in appearance. Soon, however, the leaf begins to turn paler or yellowish and finally wilts and dries. The dead tops soon become covered with a secondary mold which gives them a black color. If the leaves are blighted before the bulbs are mature a new growth of leaves may appear. These leaves may continue to develop during the remainder of the season or if conditions are favorable they, too, may be invaded by the blight fungus and killed. In this case the bulb has no chance to mature properly. In some cases according to Murphy and McKay (7) the mycelium may invade the bulb itself, in which case the outer layers of the bulb may be somewhat rough or corrugated, or the whole bulb may finally become soft.

**Morphology and life cycle of the fungus.** — This fungus has the intercellular mycelium and haustoria typical of the downy-mildew fungi. The hyphae are non-septate and the haustoria are long, slender, often ramified, tendril-like branches which penetrate the cell wall and form close contact with the cell contents. The reproductive organs are of two kinds, asexual and sexual, which are characteristic of this family of the Phycomycetes.

*Asexual or conidial stage.* — This consists of conidiophores and conidia. The former arise from the intercellular mycelium and emerge through the stomata (Fig. 47 A). They are much branched, resembling a tree with the deliquescent type of branching. The conidiophores are 300–700  $\mu$  in height and 12–15  $\mu$  in diameter of the main stalk. The conidia are elliptical in shape and measure about  $44\text{--}52 \times 22\text{--}26 \mu$ . They are borne singly on the ends of the ultimate branches of the conidiophore. The conidia germinate directly by germ tubes. It is the enormous numbers of the conidiophores and conidia emerging through the numerous stomata on the leaf that give the furry or downy appearance mentioned above under Symptoms.

*Sexual stage.* — The sexual reproductive organs consist of an oogonium



and an antheridium (Fig. 47 B). These organs arise from the inter-cellular mycelium and are imbedded in the host tissues. Sections of badly diseased onion leaves will often disclose numerous oögonia or oöspores scattered in groups among the host cells. Both oögonia and antheridia occur as swellings on the end of short branches. The oögonium is globose in shape while the antheridium is more elongated or club shaped. The central, denser region of the protoplasmic contents of the oögonium is the egg. A small tube grows out from the antheridium and penetrates the wall of the oögonium. The male nucleus from the

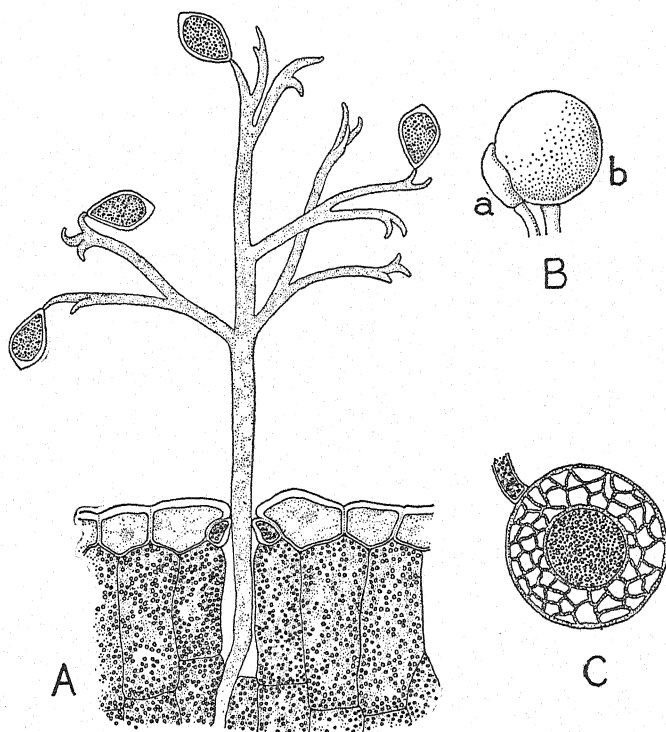


FIG. 47. — *Peronospora schleideni*. A, section of leaf showing a conidiophore protruding through a stoma and bearing several conidia. B, sex organs of the fungus; (a) antheridium; (b) oögonium. C, oöspore within the oögonium.

antheridium enters the oögonium through this fertilizing tube and unites with the egg. After fertilization occurs in this manner the fertilized egg develops into a mature oöspore (Fig. 47 C). The oöspores average about  $30\ \mu$  in diameter.

It has been the general opinion that this fungus overwinters by means



of the sexual stage in the old dead leaves. Recently Murphy and McKay (7) have stated that in Ireland oöspores are scarce and apparently play a minor part in the hibernation of the fungus. They have discovered that in that country, at least, the fungus lives as perennial mycelium in the bulbs of those varieties which are propagated by means of sets. When new leaves develop from these bulbs the mycelium extends into these leaves and conidiospores are soon produced which serve to spread the disease during the growing season. Inasmuch as the conidia are largely wind disseminated and, under humid conditions, infection readily occurs, an epidemic may break out very quickly. Bulbs are not infected directly but only by the mycelium spreading downward from the leaves which have been infected by conidia. Perhaps further investigation will show that here, as well as in Ireland, the oöspores are not the chief means of overwintering.

**Control.** — In so far as the onion mildew fungus overwinters in the old onion refuse, the destruction of this plant débris and a system of crop rotation should be practiced. Where perennial mycelium occurs in the bulbs only sets known to be free from infection should be used. Numerous attempts have been made to protect onions from infection by means of sprays. This method of control is not satisfactory in every respect yet a certain amount of protection may be had if spraying is begun in time. Bordeaux mixture 4-4-50 is usually recommended. Since the onion leaves are very waxy and smooth the use of a good sticker and spreader is absolutely necessary in order to cover the leaves well with the spray. Resin fish-oil soap or a casein spreader may be used.

#### LABORATORY STUDY

**Symptoms.** — Examine diseased leaves showing various stages of the disease. Note the growth of conidiophores and conidia on the surface. Observe leaves in the more advanced stages. Note the yellowing and finally the complete death of the leaves. Note the presence of the secondary black-colored mold in advanced stages. If invaded bulbs are available, observe the effects of the disease on them. Make drawings to show any symptoms observed.

**Morphology of the fungus.** — In sections of leaves or bulb scales find the mycelium. Can you see the haustoria entering the cells?

1. *Conidial stage.* — In good sections conidiophores can easily be found protruding through the stomata. Draw to show stoma and whole conidiophore with conidia. If sections are not available scrape off some conidiophores and conidia and mount in water.

2. *Sexual stage.* — For the study of this stage, sections of leaves are necessary. Find oögonia and antheridia, if present. Are there any mature oöspores in the section? Can you distinguish the oögonium, before fertilization has occurred, from the mature oöspores? The latter are dark brown with heavy walls and are usually

still surrounded by the old oogonial wall as if in a spherical cell having a transparent wall.

Notes. — Write an account of this disease following the outline on page 152 but modifying it if necessary to fit this case.

# REVIEW QUESTIONS

1. Describe the symptoms and signs of the onion downy-mildew disease.
2. What characteristics of the causal fungus identify it as belonging to the downy-mildew family?
3. In what form is the organism disseminated during the growing season?
4. What two means of overwintering are possible?
5. In case of bulb invasion how does the fungus get into the bulb? In what form may the fungus be found in bulbs?
6. In the tissues of what part of the plant have oöspores been found?
7. Where do the conidiophores emerge?
8. If the fungus overwinters as oöspores in dead leaves, what control measures apply especially to this manner of perpetuation?
9. If the fungus overwinters as mycelium in the bulbs, what control measures should be recommended from this standpoint?
10. What control measure is particularly appropriate to guard against dissemination by conidia during the growing season?
11. If reference number 7 is available, discuss the work done on perennial mycelium of onion mildew by Murphy and McKay.

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### Downy-mildew of Grapes

Caused by *Plasmopara viticola* (B. and C.) Berl. and De Toni

This is one of the best known of the many diseases caused by species of the downy-mildew family (Peronosporaceae). It should be clearly distinguished from the powdery-mildew (see p. 309), which is also very commonly found on grapes. Either form may appear alone in a particular region, or both may occur together in the same vicinity. In the downy-mildews the mycelium penetrates deeply into the host tissues. This characteristic distinguishes the downy-mildews from the powdery-mildews, since in the latter the mycelium as a rule penetrates only the epidermal cells.

**History and distribution.** — The best information available indicates that the grape downy-mildew is native to North America. It was first reported in the United States in 1834. The disease is common in the Mississippi valley and eastward, and it has been reported from Texas and California also. The first record of its occurrence in Europe was made in 1878. The grape Phylloxera had been previously introduced into France and in order to combat this pest more effectively the French imported some American grape stock which is more resistant to the Phylloxera. But in doing so they unwittingly brought in the mildew fungus and thus doubled their troubles. The downy-mildew has now spread all over Europe. An interesting historical fact in this connection has to do with the accidental discovery of bordeaux mixture as a fungicide for this disease shortly after the mildew became troublesome in France. The story is told that it was customary to sprinkle a mixture of bluestone (copper sulfate) and lime on the grape vines bordering the highways in order to discourage pilfering of the grapes. It was noticed that vines so treated were comparatively free from mildew. This led to experiments by the French plant pathologist, Millardet, in which he perfected the bordeaux mixture as a spray for this disease. Since that time (1883), this material has become a standard spray for many other plant diseases. As recently as 1917 the downy-mildew of grape was introduced from Europe into Australia where it has now become well established.

**Hosts.** — The disease is confined largely to species and varieties of grapes although it has been reported also on the five-leaved ivy (*Ampelopsis*). Both wild and cultivated grapes are attacked. No variety shows extreme resistance but the American grapes in general are not nearly so susceptible as the European varieties. This accounts for the greater losses suffered in Europe. This disease offers one of the many good illustrations of the fact that an introduced disease is frequently

more virulent on the new varieties of its host plant with which it comes in contact in the new country than on its host varieties in its native habitat. This is usually explained by saying that an equilibrium had been reached between host and parasite due to long association and the survival of the fittest, and that this equilibrium is broken down when the parasite is introduced to a new set of host varieties. (See Chapter IX.)

**Symptoms.** — The disease attacks all green parts of the plant, leaves, petioles, tendrils, green shoots and fruits. On any part of the plant the characteristic sign at the height of the fungus development is the white downy growth over the surface of the diseased parts.

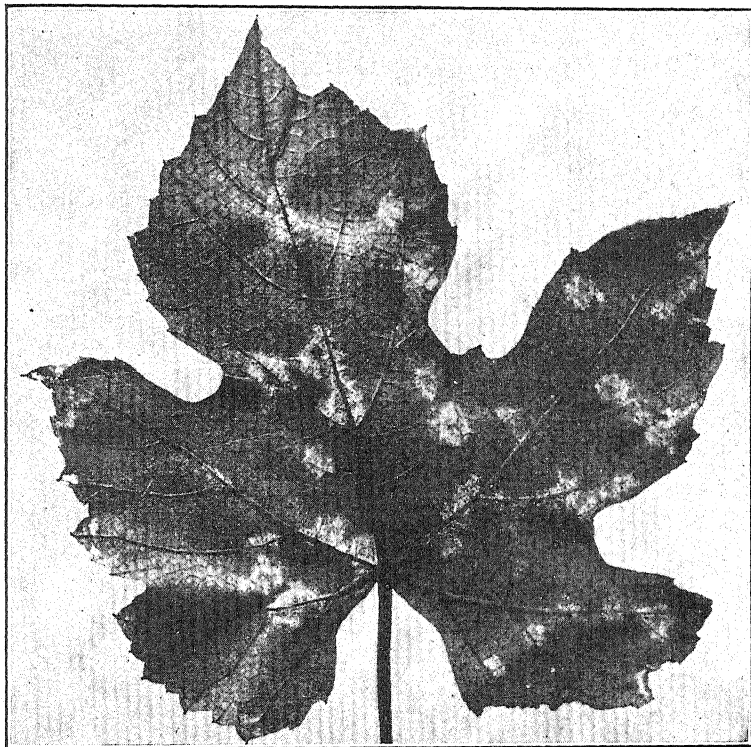


FIG. 48. — Grape leaf showing the whitish patches of downy-mildew. (Photograph from Dept. of Plant Pathology, Cornell University.)

*On the leaves.* — The mildew spots on the under side of the leaf may be of any number and size (Fig. 48). Sometimes a number of small spots appear. Again there may be only a few spots, large, or small, and

in extreme cases the whole under surface of the leaf may be covered with mildew. On the upper side of the leaf yellowish or brownish spots appear, exactly corresponding with the patches of mildew on the under side of the leaf. When examining the plant for mildew these discolored spots on the upper side of the leaf are first noticed and serve as an in-

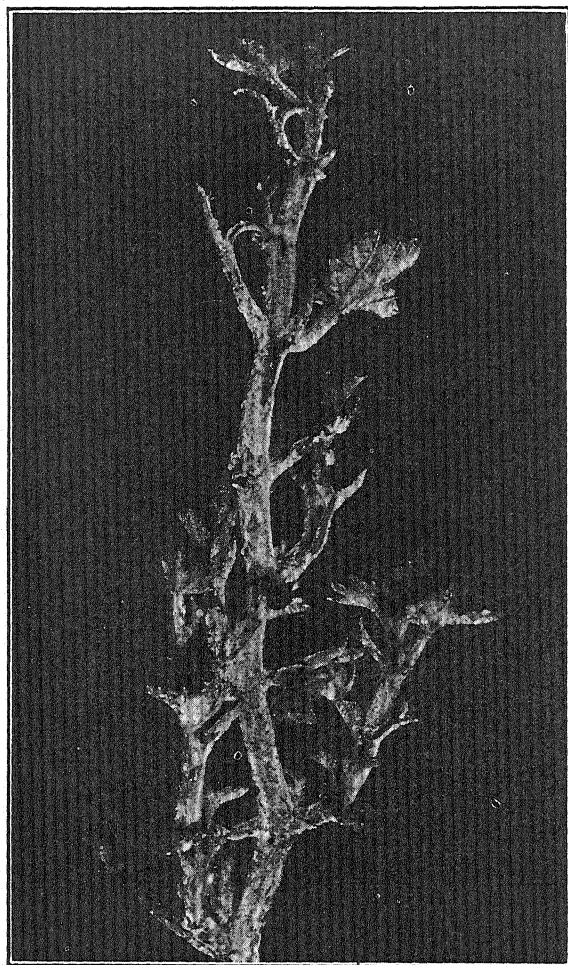


FIG. 49. — A young shoot of grape infested with the downy-mildew fungus. (Photograph from Dept. of Plant Pathology, Cornell University.)

indicator. If the trouble is mildew the fact will be disclosed by looking on the under side of the leaf immediately opposite the spot showing on top, where the growth of mildew will be found. These spots on the

leaf begin as small, indefinite, greenish-yellow areas and the discoloration gradually becomes more distinct until the typical symptoms appear. The spots finally die and become brown and dry.

*On shoots.* — The first sign is a watersoaked appearance and a slight swelling. Soon the mildew covers the lesion as is the case on the leaves. Whole shoots may be dwarfed, the leaves remaining very small and the whole densely covered with the mildew (Fig. 49).

*On fruits.* — When the disease first appears on the fruit it changes to a grayish or lead-color. There is also a hardening of the berry. Then the mildew appears over the surface (Fig. 50). Finally the fruits turn brown or red and shrivel up, ultimately becoming mummified. Shelling is a frequent accompaniment.

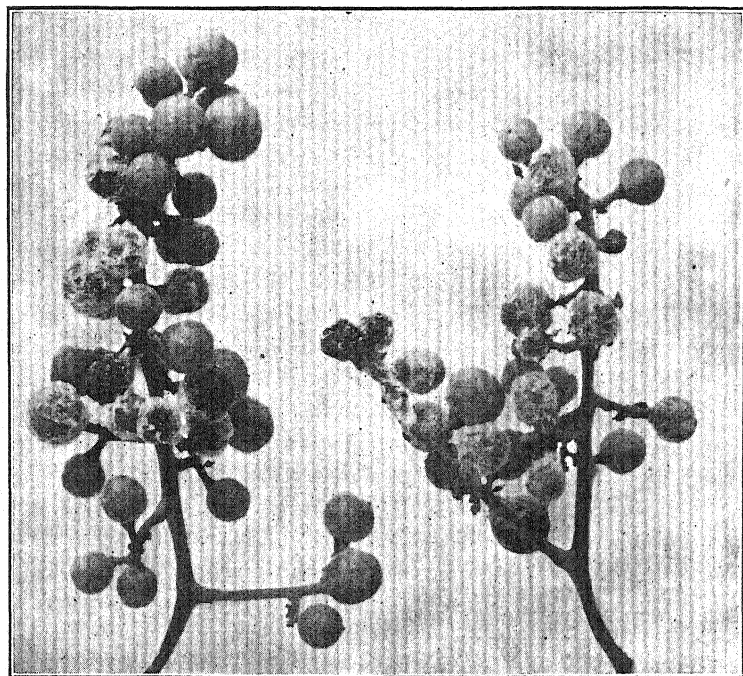


FIG. 50. — A cluster of grapes showing the effects of downy-mildew. (Photograph from Dept. of Plant Pathology, Cornell University.)

**Losses.** — Three types of injury may result from downy-mildew: (a) defoliation; (b) dwarfing and killing of the canes; and (c) destruction of the fruit. In Europe the loss is said to be very great due to the greater susceptibility of the varieties grown there. In the past severe losses have been reported from some sections of the United States. It

has been estimated that as much as 25 to 75 per cent of the crop is sometimes destroyed by downy-mildew. Reports of the Plant Disease Survey in recent years do not indicate that grape downy-mildew is of very great economic importance in the United States. In 1921 downy-mildew was reported from a majority of the states east of the Mississippi river but apparently was not considered a serious disease. In 1922

it was quite generally prevalent as usual but was reported in appreciable quantity from only a few states. Maryland reported a loss of 4 per cent, Kansas 3 per cent, New York .5 per cent, and a trace was reported from Illinois and Iowa. In 1923 the reduction in yield due to the mildew was reported from Maryland at 4 per cent, Illinois 2 per cent, Iowa 2 per cent, South Carolina 1 per cent, and New York, Pennsylvania, Minnesota and Alabama a trace.

**Morphology of the fungus.** — The vegetative mycelium is non-septate and intercellular with knob-shaped haustoria penetrating the cells. This fungus has two reproductive stages in common with most of the downy-mildews. These are the asexual or conidial stage and the sexual stage in which oöspores are produced. The conidiophores arise from the hyphae in the intercellular spaces just beneath the lower epidermis and emerge through the stomata (Fig. 51). They are characteristically branched, the primary branches arising alternately from

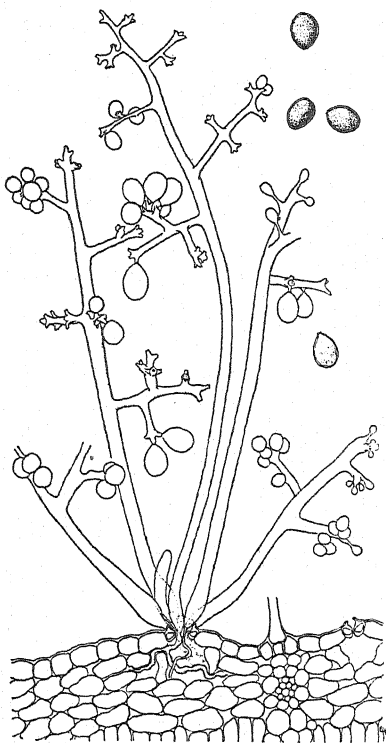


FIG. 51. — *Plasmopara viticola*. Conidiophores emerging through a stoma, and bearing conidia in various stages of development.

the trunk of the conidiophore and projecting out approximately at right angles. These branches in turn give off other branches more or less at right angles so that the system of branches shows many small branches with arms projecting out in cross-like fashion. The tips of the branches are provided with short finger-like projections on which the conidia (zoösporangia) are borne. The conidia are egg-shaped to elliptical, varying in size, the range being  $9-12 \times 12-30 \mu$ . The conidia



are really zoösporangia since in germination they give rise to zoöspores (Figs. 52, 53). The profuse growth of conidiophores and conidia on the surface of the host accounts for the downy appearance which characterizes the downy-mildews. The oöspores are produced from the mycelium imbedded within the leaf tissue. Oögonia and antheridia are formed and sex fusions occur after which the eggs become oöspores (Fig. 54). In germinating, an oöspore sends up a short unbranched stalk, at the apex of which a single conidium is produced (Fig. 55). This conidium resembles in all details the conidia produced on the ordinary conidiophores on the surface of the leaf during the summer season, and germinates by producing zoöspores in exactly the same manner.

**Life cycle.** — The fungus hibernates in the form of oöspores on the dead and fallen leaves. In the spring the leaves have decayed and partly disintegrated. The oöspores then germinate as indicated above and either the zoösporangia or the escaped zoöspores are splashed onto the lower leaves of the vines by rains. Here the zoöspores germinate in films of water and send germ tubes through the stomata. After the mycelium has established itself conidiophores are sent to the surface and conidia are produced, which serve for rapid dissemination during the summer. The conidia are transported from plant to plant and from leaf to leaf probably by both wind and rain. Water is always necessary for germination, however. The fungus grows best at a temperature of 77° to 82° F. Prolonged warm wet weather is conducive to an epidemic of downy-mildew. Dry weather holds the fungus in check although not necessarily killing it.

**Control.** — There are two possible methods of combating this disease, namely, (a) sanitation and (b) spraying. Theoretically the destruction or plowing under of the old leaves should eradicate the source of inoculum since the fungus hibernates as oöspores in the fallen leaves. Practically the chief method in use is the application of a protective spray to the susceptible parts. Bordeaux mixture, 5-5-50, is usually recommended. The first spray should be applied just before the blossom buds open and a total of six applications should be made at intervals of two weeks.

#### LABORATORY STUDY OF GRAPE DOWNY-MILDEW

**A. Symptoms.** — Observe the symptoms of this disease on leaves, shoots and fruits.

**Leaves.** — Note the distribution of the mildew over the leaf surface. Which surface shows the downy growth? What symptoms appear on the opposite surface? Of what does the downy growth consist? Sketch to show appearance of both sides of the leaf.

**Shoots.** — At what age are shoots liable to attack? Compare the symptoms with those on leaves.



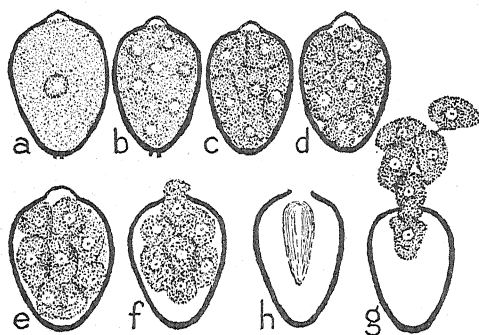


FIG. 52. — Germinating conidia (zoösporangia) of *Plasmopara viticola*. c, d, protoplast dividing; e, eight zoöspores ready to emerge; f, g, zoöspores emerging from zoösporangium. (After Gregory, *Phytopath.* 2:235-249.)

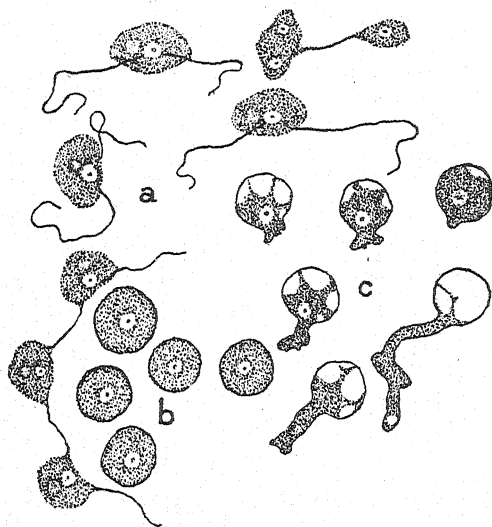


FIG. 53. — Zoöspores (swarmspores) of *Plasmopara viticola* after escaping from the conidium. a, motile spores; b, quiescent stage; c, germination of swarmspores. (After Gregory.)

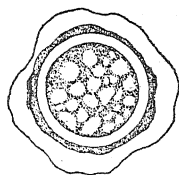


FIG. 54. — Oöspore of *Plasmopara viticola* contained within the old oögonium. (After Gregory.)

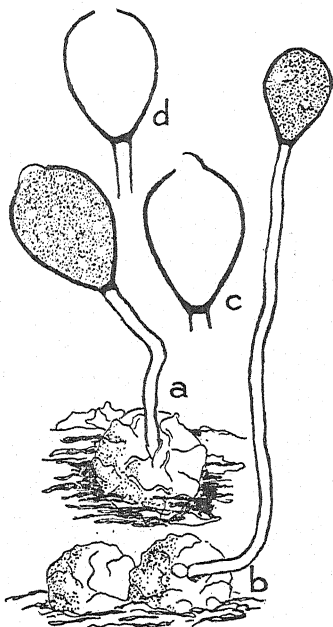


FIG. 55. — Germination of oöspores of *Plasmopara viticola*. a, conidiophore arising from oöspore and bearing a mature conidium; b, an immature conidium; c, d, empty conidia after germination. These conidia germinate in exactly the same manner as those borne on the branched conidiophores in the regular imperfect stage of the fungus. (After Gregory.)

*Fruits.* — The young fruits are frequently attacked. Observe the surface indications of the disease. What is the ultimate effect upon the fruit?

**B. Morphology and life cycle of the fungus.** — This fungus is typical of the downy-mildew family in both its vegetative and reproductive phases. Examine sections of diseased leaf and try to find the mycelium of the fungus. Is it superficial or does it penetrate deeply into the tissues? Compare with the mycelium of the powdery mildews in this respect (see Chapter XVIII). Try to find the haustoria.

*Asexual stage.* — Examine sections of a mildewed leaf, or mount some of the downy growth from the under side of the leaf. In the sections note the emergence of the conidiophores. Through what do they emerge? **Draw** a conidiophore to show the characteristic branching. Compare with the conidiophores of other downy-mildews described in this text. Find out how the conidia germinate. Are they true zoösporangia? Compare with the germination of conidia of the onion downy-mildew and of the late blight of potato. **Draw** conidia. What part does the asexual stage play in the life history of the fungus?

*Sexual stage.* — Examine sections of dead leaves picked up from the ground in the early spring. Find the oögonia and antheridia or the mature oöspores imbedded in the dead leaf tissue. **Draw.** What part do the oöspores play in the life history of the fungus? Describe the germination of the oöspore (see reference 3). Describe the complete life history of the fungus including overwintering, primary infections, summer dissemination, and intensification of the disease.

**C. Notes.** — Write full notes on the grape downy-mildew disease, following the outline on page 152.

#### REVIEW QUESTIONS

1. Describe the symptoms and signs of downy-mildew on all parts of the grape plant.
2. In general what varieties of grapes are most susceptible to this disease?
3. Describe the vegetative structure of the causal fungus. Is the mycelium inter-cellular or intra-cellular? How does it make contact with the host cell in securing its food supply?
4. Is the type of haustorium found in this fungus typical of all downy-mildews?
5. What is the difference between a true conidiospore and a zoösporangium such as occurs in the life history of this fungus? (Compare with the onion downy-mildew fungus.)
6. What part do the dead fallen leaves play in the life history and control of the grape downy-mildew fungus?
7. What two control measures are to be recommended based respectively on the method of overwintering, and on the means of summer spread?

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### Late-blight of Potato

Caused by *Phytophthora infestans* (Montagne) De Bary

The term late-blight is the one usually applied to this disease although it is sometimes called downy-mildew, black-blight, potato-blight, or simply blight. The disease on the tubers is properly referred to as late-blight rot. It is, or was, the most feared of all potato diseases. This is true in some regions, at least, because of the well-known epidemics which sweep over certain sections at irregular and unpredictable intervals. Since control methods are now better understood, it is possible to ward off such disastrous results as formerly followed outbreaks of this disease.

**Historical.** — It is difficult to trace with certainty the history of this disease but it seems more than probable that it originated in South America, the native home of the potato plant. The potato itself was first introduced into Europe probably some time during the sixteenth century. Its culture spread until during the latter part of the eighteenth century and the early part of the nineteenth century it was grown extensively throughout Europe and America and formed one of the principal food crops for many people. This was especially true in Ireland. It has been thought by some that the late-blight fungus may have been introduced along with the early importations of potatoes from South America. There is evidence that a disease of potatoes which might have been late-blight existed in the Andes Mountains of South America as early as 1571. There is no definite proof, however, that late-blight was present in Europe prior to 1830-1840. It seems probable that the disease was not introduced on the earliest importations of potatoes but came into Europe on subsequent importations, probably not much later than 1830. There are several references to it in the writings of German, French, Belgian, Danish and British investigators appearing between the years 1840 and 1845. In the latter year the famous Irish famine occurred, due to the failure of the potato crop because of an epidemic of late-blight. It is probable that the disease was introduced into the United States and Canada at about the same time as the European introduction.

Since 1845 investigations on the cause and control of this disease

have been pursued with vigor, first in Europe, and later in the United States. The cause of the disease was first ascribed to a parasitic fungus by Von Martius in 1845. The fungus was described by Montagne (15) in 1845 and named *Botrytis infestans*. In 1876 the fungus was given its present name, *Phytophthora infestans*, by De Bary (5) who also did some very good work (3, 4) on the parasitism of the organism and its causal relation to the late-blight disease. Jensen carried out extensive investigations on tuber infection and remedial measures. In the United States, Clinton, L. R. Jones and his associates, Orton, Melhus, and many others have continued the researches to the present time.

**Geographic distribution.** — Late-blight is now known in all potato-growing sections of the world. However, it does not assume great importance in all regions. In localities where the growing season is uniformly dry and hot there is comparatively little late-blight. On the other hand a cool, humid climate favors the disease. In the United States it is most likely to be severe in that section north of the Ohio river and east of the Mississippi, especially in New York and northern New England. It occurs occasionally also along the Pacific coast. Late-blight is often quite prevalent in south-eastern Canada. Great Britain and Ireland frequently suffer enormous losses from this disease. It is generally distributed throughout the northern countries of Europe, and of course it occurs in South America. In any country or region the factor that favors the occurrence of the disease in harmful amounts is the prevalence of cool, wet weather during the middle and latter part of the growing season.

**Hosts and varietal susceptibility.** — While the potato (*Solanum tuberosum*) is usually the most common host of *Phytophthora infestans* it is by no means the only species attacked. It is frequently found on the tomato and it is reported that sometimes it may become as destructive to this host as to the potato. It has been reported on several other species of *Solanum* and on *Petunia*. All of the above hosts belong to the nightshade family (*Solanaceae*), but it is reported on a few species of plants not belonging to this family. However, it is best known as a potato disease and its occurrence on other species is of relatively little importance as compared with its effects on the potato crop.

Not all potato varieties are equally susceptible to late-blight. In Europe, in the early days of late-blight investigations, resistant varieties were developed. In the United States these foreign resistant types have not proved popular. In this country some of our leading commercial varieties, such as Early Ohio, Irish Cobbler, Green Mountain, Rural New Yorker and Pearl are very susceptible. On the whole, the development of blight-resistant varieties of potatoes which are at the

same time most desirable from the commercial standpoint has not reached a high state of perfection in the United States up to the present time. There is reason to believe, however, that this will finally be accomplished.



FIG. 56. — Effects of late-blight of potato in the field. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

**Economic importance.** — The losses due to late-blight are of two kinds, actual reduction in yield due to attacks on vines and foliage, and damage to tubers when invaded by late-blight rot. If the vines are blighted down before the crop is mature, of course the tubers are prevented from reaching normal size and the yield is reduced proportionately. Sometimes great loss is sustained on account of direct attack on the tubers themselves. The estimates given below include losses from both types of injury. In those regions subject to severe outbreaks of late-blight it is not unusual for growers to suffer losses of a large percentage of the crop. A decrease in yield of 50 to 100 or more bushels per acre is not uncommon. In 1912 the loss in New York alone was estimated (2) at \$10,000,000. In 1921 the loss in the United States as a whole was estimated (1) at 0.5 per cent of the crop, or 2,106,000 bushels; in 1922 the total loss was 5 per cent, or 11,288,000 bushels; in 1923, 0.1 per cent, or 623,000 bushels; and in 1924, 3.9 per cent, or

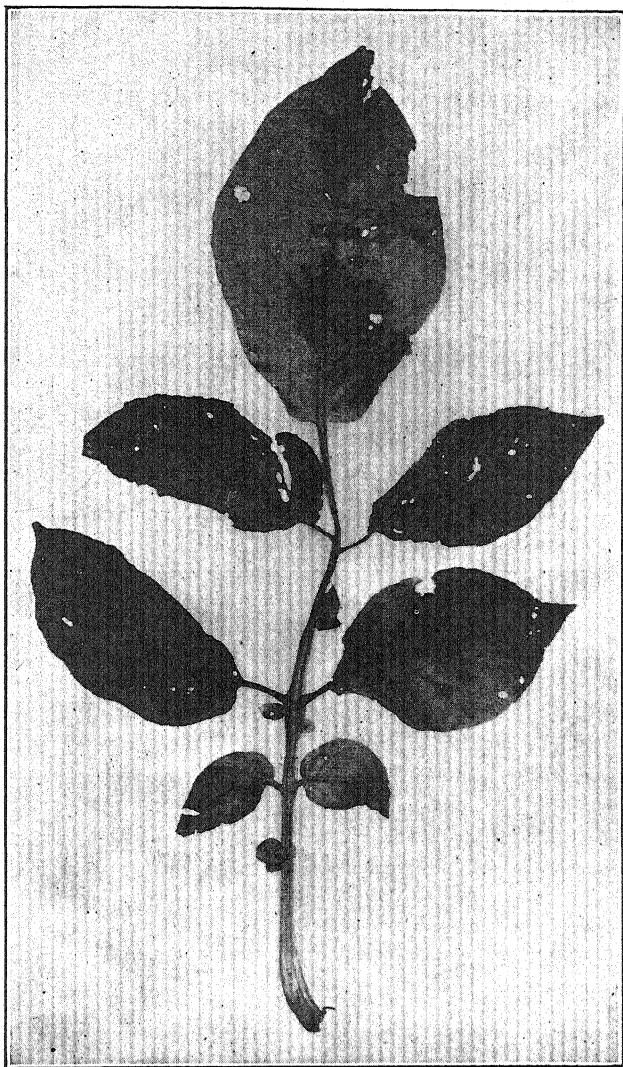


FIG. 57. — Potato leaf showing effects of late-blight. The darkened areas have been invaded by the fungus. The perforations are the work of insects and have no relation to the late-blight disease.

21,980,000 bushels. The classical example of the devastation which this fungus is capable of causing is that of the total failure of the potato crop in Ireland in 1845 and the resulting famine.

**Symptoms.** — The effects of this disease are manifest on both the aerial and the underground parts of the plant. The symptoms on leaves, stems, petioles and pedicels are all similar in character. The first indi-

cation is a watersoaked appearance usually at the edge or tip of the leaflet. These infected areas enlarge more or less and may involve the entire leaflet if weather conditions are favorable. The diseased area

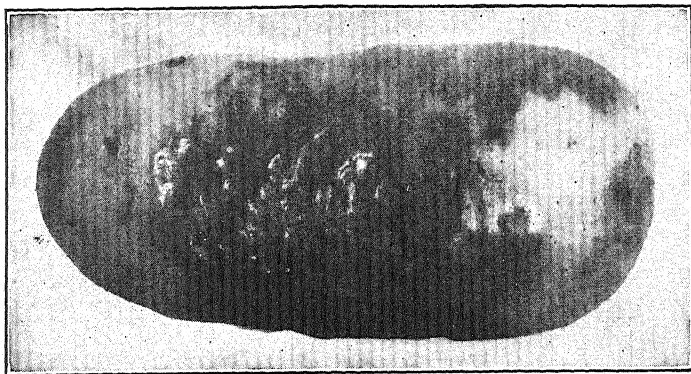


FIG. 58. — Potato tuber showing surface appearance of late-blight rot. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

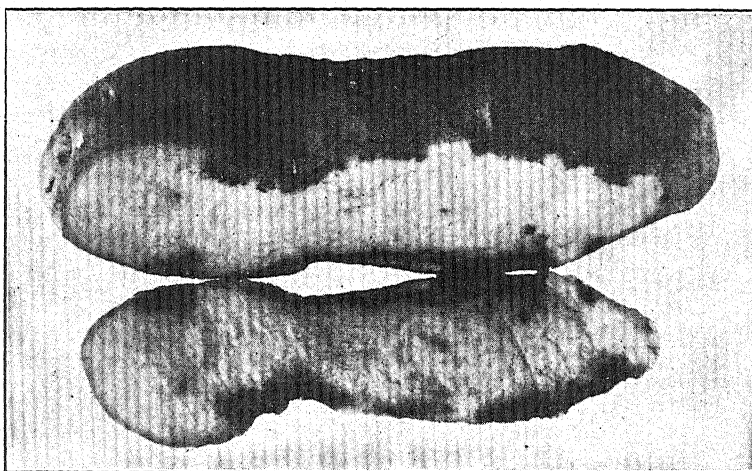


FIG. 59. — Section of potato tuber showing the late-blight rot penetrating to a depth of several millimeters in an irregular manner. (After McKay, Ore. Agr. Exp. Sta. Cir. 24.)

turns darker in color (Fig. 57) and may become dry, blackened and shriveled if the weather becomes drier. In continued wet weather, the leaves and vines may completely rot. Other signs consist of the downy growth of conidiophores which comes to the surface on the under side of the leaf giving it a white, moldy appearance. This downy growth is characteristic of the downy-mildews. In moist weather the rotting foliage emits a characteristic odor which is a helpful diagnostic character



in identifying the disease. The rot on the tubers is characterized by the slightly shrunken and darkened appearance of the diseased areas (Fig. 58). The lesions begin as small spots and enlarge until sometimes a large part, or even the entire surface, of the tuber is involved. Unless invasion by secondary saprophytes occurs, the late-blight rot is a shallow rot penetrating to a depth of only a few millimeters (Fig. 59). The rotted tissue presents a dry, granular, brown or brick-red appearance. It is sometimes called dry-rot but this name has been reserved for another specific potato disease.

**Morphology of the fungus.** — *Phytophthora infestans* belongs to the class Phycomycetes, hence the vegetative mycelium is non-septate. Like that of the other downy-mildews the mycelium is intercellular in the leaf tissues. The hyphae send small filamentous haustoria into the cell cavities. Two forms of reproductive bodies are produced by the fungus, conidia and oöspores, although it has not been definitely established that the latter are functional.

**Conidial stage.** — Conidiophores emerge singly or in groups of two to four through the stomata. The conidiophores are sparingly and characteristically branched. They present a series of swollen joints on both the main stem and the branches. The conidia (zoösporangia) are somewhat eggshaped and are produced on the tips of the branches (Fig. 60). When the conidium is mature it is pushed aside and the branch continues growth and produces a new conidium at its tip. In this way several conidia may be produced on each branch, the swollen places marking the points at which conidia were borne. Conidia germinate in two ways: (a) directly by means of germ tubes, or (b) indirectly by dividing up internally into several zoöspores (Fig. 61). Environmental conditions appear to determine the method of germination. Temperature seems to be at least one of the factors. In general low temperatures, 10° to 20° C., seem to favor zoöspore production while higher temperatures, 25° C., favor direct germination. If zoöspores are produced they can cause direct infection by means of germ tubes. If conidia germinate directly their germ tubes may cause infection so that zoöspore production is not necessary for infection.

**Oöspores.** — Many of the downy-mildews (Peronosporales) are known to produce functional resting spores (oöspores) and this has led to the supposition that the late-blight fungus also may reproduce in this manner. Persistent search has failed to furnish conclusive evidence on this point. A few investigators claim to have found them in potato leaves but others have failed to substantiate these claims. Jones and others (8) have found oöspore-like bodies in pure cultures of the fungus but produced no conclusive evidence that these bodies are functional. The most



recent evidence has been produced by Murphy (23). He found oögonia and parthenogenetic resting-spores on portions of potato tubers in impure culture. Occasionally he found a complete set of organs in-

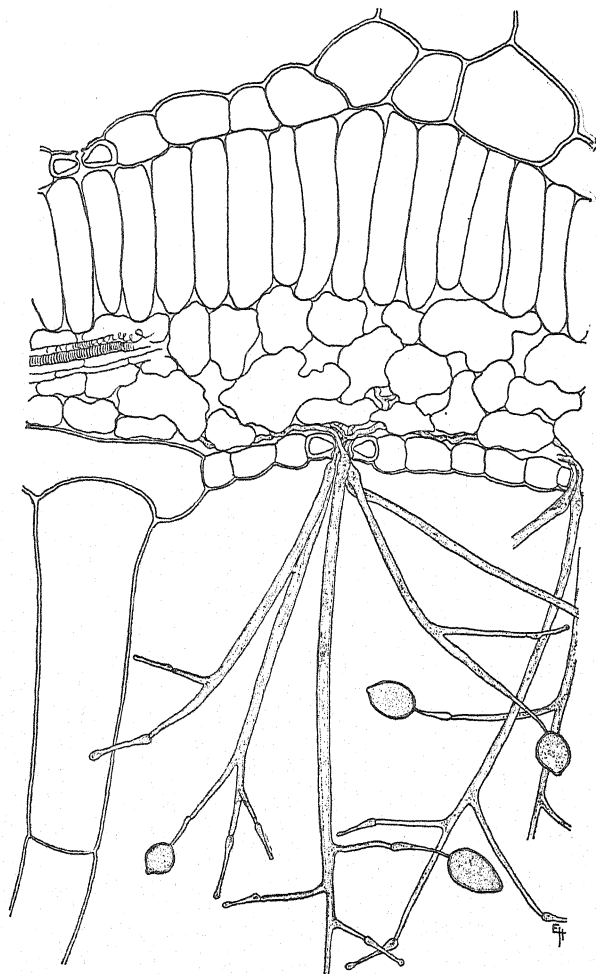


FIG. 60. — *Phytophthora infestans*. Section of potato leaf showing several conidiophores protruding from stomata.

cluding oögonium, antheridium and oöspore in pure culture on pieces of potato tuber. Later oögonia and parthenogenetic spores were found on the surface of a cut tuber in the soil. There is as yet, however, no definite proof that these resting-bodies are of any great importance in the life history of the fungus.

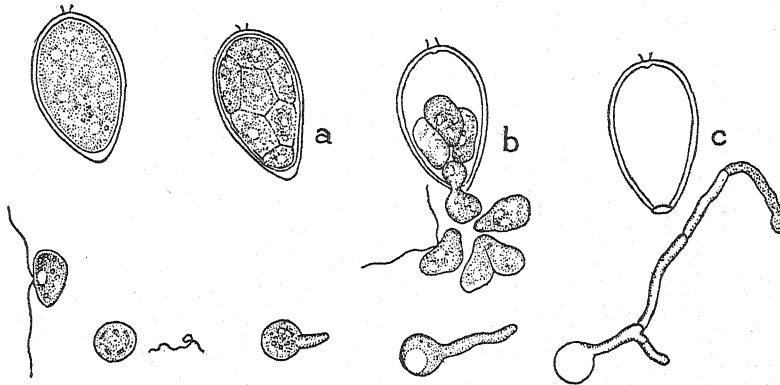


FIG. 61. — Germinating conidia of *Phytophthora infestans*. a, protoplast of conidium (zoösporangium) divided into eight cells; b, zoöspores emerging from conidium; c, empty zoösporangium. Below: various stages in the germination of a zoöspore. (Redrawn after Ward.)

**Life cycle.** — At present the preponderance of evidence points to the conclusion that the fungus hibernates as mycelium in potato tubers. The fungus will produce conidia on the cut surface of diseased tubers. When diseased tubers are planted the fungus sometimes grows up in the sprouting shoots until it reaches the surface of the ground where conidia are produced. These conidia are carried by wind, rain or possibly insects to nearby potato leaves and thus serve to initiate an epidemic if weather conditions are favorable. It takes only a few days, sometimes less than a week, after infection for a crop of conidia to appear, so that a very few diseased tubers in the seed lot may suffice to cause serious damage.

**Tuber infection.** — The tubers are infected by spores and not by the mycelium growing down the stem until it reaches the tubers. Tubers may become inoculated by spores which have been washed into the soil by rains or they may become infected after digging by coming in contact with spores from diseased foliage. In storage the fungus may fruit on the surface of diseased tubers and thus spread to other tubers, but this can occur only under the most favorable conditions for the growth of the fungus. When the potatoes are stored under proper conditions little if any tuber infection is likely to occur in storage.

**The weather and late-blight.** — It has long been known that weather conditions play an important part in late-blight epidemics. As early as 1888 (8) it was stated that serious outbreaks of late-blight always occur in seasons when the humidity is high and the temperature fairly low (65° to 75° F.). In Maine (16) the years 1907 and 1909 witnessed severe outbreaks and during both these seasons the weather was most favorable for the development of the disease. Studies made in Vermont

(10) over a long period of years indicate in a general way that wet years are apt to be bad late-blight years, though there are exceptions to this rule which can be accounted for only by taking other factors such as temperature, sunshine and winds into consideration. Pure culture studies of the fungus (8) indicate that the best growth occurs when the cultures are held between 16° and 19° C. Below 16° the growth is slower and below 5° no growth occurs. No growth occurs at or above 30°. Studies at Wisconsin (13) showed that the optimum temperature for indirect or zoospore germination in water is 12°–13° C., with a minimum of 2°–3° C., and a maximum of 24°–25° C. For direct germination of conidia by germ tubes higher temperatures are required with an optimum of about 24° C. Conditions favorable for spore germination are also optimum for host infection. There have been at least three epidemics of late-blight in Iowa (7), occurring in 1885, 1903 and 1915. Studies of weather conditions during these years show, in all three years, that the rainfall for July was far above normal and, in the first two years, August was also a wet month. Subnormal temperatures also prevailed for the most part during these periods. In New Jersey (12) weather conditions were correlated with outbreaks of late-blight, the results showing that over a period of 34 years the rainfall for the months of June, July, August and September was above the average in 16 of the 34 years and that blight was prevalent in 13 of the 16 years. A similar correlation of temperatures indicated 18 years of subnormal temperatures, during 12 of which blight was reported.

**Control.** — The following items must be considered in devising control measures for late-blight: (a) The fungus hibernates as mycelium in tubers. (b) It comes to the surface of the ground on young shoots from diseased tubers, and sporulates. (c) The conidia are scattered far and wide, by wind and rain, both from the primary infections and from the more numerous secondary infections. (d) The new tubers are infected by spores carried down into the ground by rains, and by spores which get on the tubers from diseased foliage after digging.

Based on these facts there are three major items to be observed in combating the disease. First, use seed free from hibernating mycelium. Since there is no method of treating the seed tubers to rid them of the fungus, certified seed, or seed known to be disease-free must be used. Second, if any disease appears in the field the only means of holding it in check is by spraying. Careful growers in regions subject to blight spray regularly every year as insurance against loss. The spray acts as a protection against infection; hence if one waits until the disease appears in the field it may be too late to save the crop, especially if the weather is optimum for an outbreak. The standard spray for late-

blight is bordeaux 5-5-50. Other formulas are sometimes used, as 4-4-50, 6-6-50, 6-10-50, etc. The spraying is usually begun when the vines are about six inches high and at least four applications are made at intervals of a few days to two weeks. If weather conditions continue favorable for infection it may be necessary to increase the number of applications and shorten the interval. During recent years, copper-lime dust has been used to some extent instead of bordeaux, but in general it does not give as satisfactory results as the liquid spray (20). It has been fairly definitely determined that spraying potato plants with bordeaux mixture has a decidedly beneficial effect upon their general vigor and productiveness regardless of the presence of late-blight. Thus the general tonic effect upon the crop makes spraying a profitable practice even in years when no blight appears. Third, due precautions must be observed at harvest time to prevent tuber infection. If the vines have been blighted, digging should be delayed until the vines have died and dried out. Care should be taken to prevent the tubers from coming in contact with the vines. Piled tubers should never be covered with vines. Rotted tubers should be sorted out before the crop is stored. Proper storage will help materially in retarding development of tuber-rot even if there is opportunity for inoculation at digging time.

#### LABORATORY STUDY OF LATE-BLIGHT

##### A. Symptoms.

*On foliage.* — Examine diseased leaves or other above-ground parts and note the symptoms and signs in detail. Note color, shape and size of the diseased areas. On fresh material the downy growth on the under side of the leaves may be observed. Observe with the naked eye and also examine with a hand lens. Familiarize yourself with these characteristics of the disease so that you can recognize it in the field. Draw leaf to illustrate the symptoms.

*On tubers.* — Observe both the surface characters, and the internal appearance of the rot as shown in sections of rotted tubers. Note surface color and whether sunken or raised. How deep does the rot extend in sectional view? What is the texture and color of the rotted tissue? Draw tuber to show both surface characters and sectional view.

##### B. Morphology of the Fungus.

If sections of leaf or tuber showing the vegetative mycelium are available, study under the microscope and draw to show relation of the fungus to the host tissue.

*Conidial stage.* — In leaf sections find the conidiophores emerging from the stomata. Note characteristic branching and jointed or nodose appearance. Find conidia and note shape and size, also look for nuclei in the conidia. How many nuclei can you count in a single conidium? If fresh material is available, find out what the conditions for germination are and try to germinate the spores. What two types of germination are possible? Under what conditions is each type supposed to occur? Draw conidiophores and conidia. Show any germination stages available.

*Oöspores.* — If cultures showing oöspores are available, study and draw. Are the oöspores functional in the life history of this fungus? (See text and references.)

### C. Life cycle.

After studying the morphology of the fungus in detail, look up the life history in text and references. Find out how the fungus hibernates, its means of dissemination and the time, place and manner of infection, also the conditions favorable to infection and spread of the disease.

### D. Notes.

Write complete notes embodying all information gained from laboratory study and from reading.

### REVIEW

1. Describe the symptoms of late-blight of potato on all parts of the host attacked.
2. Describe the morphological characters of the fungus.
3. Describe the complete life cycle of the causal organism, including perpetuation, dissemination and infection.
4. At what season and under what conditions is an outbreak of this disease to be expected?
5. Discuss in detail the relation of weather to late-blight epidemics. (References 7, 12, 16.)
6. Discuss the evidence as to the persistence of *Phytophthora infestans* in the soil. (Reference 19.)
7. Discuss the problem of preventing tuber infection.
8. Why is seed selection more effective than seed treatment in combating late-blight?
9. Discuss dusting versus spraying for the control of this disease. (Reference 20.)
10. If a farmer sprays regularly every year as an insurance against loss from late-blight, is the premium he pays a total loss in years when no blight occurs? Why?

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## CHAPTER XVIII

### DISEASES CAUSED BY FUNGI—ASCOMYCETES

Fungi belonging to the class, Ascomycetes, are responsible for some of our worst plant diseases. The Ascomycetes are characterized by septate mycelium, and by the ascus (sac) in which the principle spore form, the ascospore, is borne. Because of the fact that the spores are borne in this sac-like structure, called an ascus (Fig. 64), the Ascomycetes are sometimes referred to as the sac-fungi. The majority of species in this class produce some kind of a fruiting body, known as an ascocarp, in which the asci and ascospores are borne. There are different types of ascocarps, such as the apothecium, or cup (Fig. 69), and the perithecium (Fig. 90). Most species of fungi belonging to the Ascomycetes possess two spore forms in the reproductive stage. In addition to ascospores, the production of which places these fungi in the class Ascomycetes, there is usually an imperfect or conidial stage which appears preceding the production of ascospores. However, in certain cases, such as the clover stem-rot fungus, there is no conidial stage, the only known spore form being the ascospores. There are several species of ascomycetes which, so far as known, produce only ascospores, but the great majority of these fungi produce both ascospores and conidiospores in their complete life cycles. In many cases species of ascomycetes spend a part of their life cycle as parasites and the remainder of the cycle as saprophytes, usually on the dead parts of plants upon which they first lived as parasites. This is well illustrated by the apple-scab fungus which invades the green leaves and produces its conidiospores while living parasitically, then continues to develop during the following winter as a saprophyte on the dead, fallen leaves, finally producing the perithecial or ascosporic stage in this saprophytic condition. In other cases, such as the clover stem-rot, the mycelium invades living plants but no conidiospores are produced. When the infested tissues die, the fungus produces sclerotia which later give rise to ascocarps and ascospores. Because of this facultative nature of the ascomycetes these fungi can generally be cultured readily on artificial media.

Examples of diseases caused by species of sac-fungi are peach leaf-curl (*Eoascus deformans*), cherry witches'-broom (*Eoascus cerasi*), plum pockets (*Eoascus pruni*), brown-rot of stone fruits (*Sclerotinia*



*fructicola*), stem-rot of clover and other legumes (*Sclerotinia trifoliorum*), apple-tree anthracnose (*Neofabraea malicorticis*), the powdery mildews (*Erysiphaceae*), ergot (*Claviceps purpurea*), European canker (*Nectria galligena*), wheat-scab (*Gibberella saubinetii*), black-rot canker (*Phylospora malorum*), take-all (*Ophiobolus cariceti*), and apple-scab (*Venturia inaequalis*). A number of these are discussed in the following pages and references are appended dealing with some of the ascomycete-caused diseases which cannot be discussed in detail here.

### Peach Leaf-curl

Caused by *Exoascus deformans* (Berk.) Fekl.

This disease is one of the well-known peach troubles which assumes great economic importance in many peach-growing regions. It is known under various names such as leaf-curl, curly-leaf, curl-leaf, curl, and leaf-blister. The Germans know it as Krauselkrankheit, and in France it is called cloque du pêcher. Most of these names indicate that it is a leaf trouble although its attacks are not confined entirely to the leaves. Twigs are quite commonly invaded by the fungus and the blossoms and fruit are sometimes attacked.

**Historical.** — The origin of peach leaf-curl is not known. It has been suspected that it came originally from the native home of the peach in central Asia but this has not been verified. It was reported from England as early as 1821, and was present in Australia in 1856. It was studied in France at least as early as 1866. The writer has been unable to find any mention of this disease in the American literature prior to 1883. In 1894 Atkinson (5) published a taxonomic study of the peach leaf-curl fungus together with several other species of *Exoascus* which attack various stone fruits including plums and cherries. In 1900 Pierce (10), published the results of his investigations on this disease, the most extensive ever made in this country. Since 1900 many other briefer articles have been published in the United States, dealing chiefly with control measures. Some studies have been made to determine the exact method of hibernation but without complete success.

**Geographic distribution.** — Peach leaf-curl is present on every one of the six continents. In North America it occurs in the United States and Canada. In South America it is known in some of the peach-growing countries, especially Chile. In Europe it is widespread in many countries on the mainland and also in the British Islands. The disease occurs both in South Africa and in North Africa along the

Mediterranean Sea. It has long been a serious peach disease in both Australia and New Zealand. Leaf-curl is reported as prevalent in both Japan and China, into the former of which it was apparently introduced on peach trees imported from America.

While the disease is more or less prevalent in all peach-growing countries its distribution is by no means uniform. It is very noticeable that leaf-curl is more prevalent in those regions not far removed from large bodies of water. In America this is illustrated in the counties bordering on the Great Lakes and in the coast regions of California and the regions west of the Cascade Mountains in the Pacific Northwest. Regions farther inland do not appear to suffer so severely, although a cool, wet spring favors the development of the disease wherever the fungus is present.

**Hosts and varietal susceptibility.** — The attacks of this particular species of *Exoascus* seem to be confined to the peach, the nectarine and the peach-almond. Other species of *Exoascus* are found on plums, cherries and other plants but *E. deformans* is apparently unable to attack fruits other than the peach and its derivatives. As is the case with other diseases there does not seem to be any hard and fast rule regarding the susceptibility of various peach varieties. Any particular variety varies in its resistance to leaf-curl from season to season and in various sections of the country. Undoubtedly ecological factors such as weather and climatic conditions play an important part in bringing about this variableness. Elberta is usually considered the most susceptible of all varieties, and Crawford one of the more resistant varieties. Yet in New York (14) in 1908 and 1909 a few growers reported Crawford as quite susceptible and Elberta as but slightly affected. Carman is said to be very susceptible while Richards is resistant. In 1923 reports from Virginia (3) indicated Elberta and Carman to be more susceptible than Early Crawford. In Illinois, during the same year, Belle of Georgia was listed as most susceptible, Elberta less so, and Champion least susceptible.

**Losses.** — It is difficult to arrive at accurate estimates of the damage done by peach leaf-curl because of the nature of the disease. While the fruits are frequently attacked directly, the injury is usually indirectly sustained through attacks on the foliage. The character of the damage done is of four or five types. The most extensive and most apparent injury of course is suffered by the leaves. In severe cases trees are partially or totally defoliated. Trees usually put out a second crop of leaves later in the summer after defoliation occurs but this saps the vitality of the tree, especially if it happens for several years in succession. Sometimes the blossoms or young fruits are attacked directly and to

this extent the set of fruit is diminished but probably the devitalizing effect of successive defoliations is more largely responsible for poor sets than these direct attacks on blossoms and fruits. Many small twigs are killed and this is more or less injurious to the tree. Especially is this true of nursery stock.

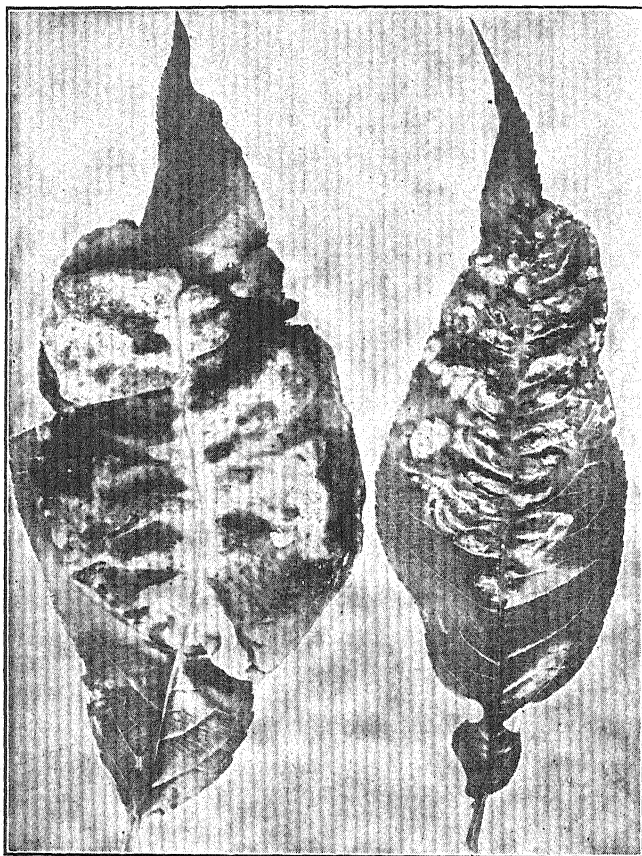


FIG. 62.— Peach leaves showing typical leaf-curl symptoms. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

Pierce (10), in 1900, estimated that the total loss in the United States due to this disease probably amounted to about \$3,000,000. In 1921 the loss in this country (1) was reported at .6 per cent or 254,000 bu., of which California lost 149,000 bu. In 1922 the total loss (2) for the United States amounted to 1.6 per cent or 1,145,000 bu. and in 1924 it was estimated (4) at 840,000 bu.

**Symptoms.** — The leaves, twigs, blossoms and fruits of the peach may be directly attacked by the leaf-curl fungus.

*On leaves.* — The first symptoms become evident shortly after the young leaves begin to emerge from the bud. Reddish colored areas appear and these areas soon show a tendency to become puckered or curled and to become thicker than the healthy areas of the same leaf or leaves which are entirely free from attack. As the disease progresses the diseased area may enlarge until the entire leaf is invaded or the fungus may remain confined to only a part of the individual leaf. Any part of a leaf may be diseased, ranging from small spots only a few square millimeters in area up to the entire leaf. As the leaf grows to maturity the diseased part becomes much thickened so that eventually it is several times as thick as the healthy part and correspondingly more rigid and brittle in texture. The parenchymatous part of the leaf is stimulated to excessive cell division which results in the thickening and expanding of the leaf blade and, since the midrib does not elongate equally, the blade consequently becomes much puckered, folded or curled. (Fig. 62.) The leaf finally shows a characteristic grayish or silvery bloom over the upper surface due to the appearance of the fruiting layer of asci which ultimately covers the entire surface of the diseased area. The leaves eventually turn yellow and die. Seriously diseased leaves usually die and drop before mid-summer.

*On twigs.* — The tips of the young shoots are frequently invaded. This results in a shortening and thickening of the new shoot and sometimes in other deformities (Fig. 63). Invaded shoots usually die. The clusters of leaves on these deformed shoots usually cling tenaciously to the shoots so that the dead twigs may be easily seen during the following winter and spring before a new crop of leaves appears. If an infested twig does not die the growing tip may continue to develop a normal shoot beyond the diseased area leaving a swollen place at the base of the year's growth. Gumming frequently occurs on the invaded parts of the twigs.

*On flowers and fruits.* — The blossoms are sometimes attacked. Invaded blossoms usually fall, thus decreasing the set of fruit. Since this phase of the disease is relatively evanescent it is seldom observed and consequently there is apparently no information available as to the amount of damage caused by blossom-curl. The young fruits are likewise subject to attack. They also usually drop early, but occasionally half-grown fruits showing the curl symptoms may be found still hanging on the tree. The symptoms show on the fruits as elevated areas with wrinkled, uneven surfaces and irregular, wavy margins. The diseased area is conspicuously and abruptly elevated above the healthy surface.



FIG. 63. — Peach shoot showing the distorting effect on the twig as well as on the leaves. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

**Morphology and life history of the fungus.** — The mycelium invades the leaf parenchyma and the cortical parenchyma of the twigs. The cells of the hyphae vary much in length, shape and size in various parts of the host tissue and in the various stages of development (10). After the vegetative mycelium has developed extensively in the leaf parenchyma, there arise, from the hyphae just beneath the upper epidermis, branches which penetrate between the epidermis and the cuticle forming a dense mass of hyphae. From this subcuticular layer numerous asci arise. Finally the cuticle is entirely lifted off and torn away, exposing

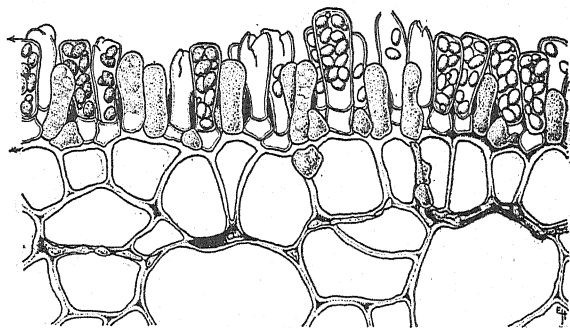


FIG. 64. — *Exoascus deformans*. Section of peach leaf showing layer of asci on the surface.

the asci which stand perpendicular to the surface and form a continuous layer over the entire diseased area (Fig. 64). It is this layer of exposed asci, after the cuticle has sloughed away, that gives the leaf its silvery appearance at this stage. The asci are somewhat club-shaped, the top being broader than the base and more or less truncated (Fig. 64). They average  $38 \times 11 \mu$  in size. The number of spores per ascus varies from 3 to 8, the latter being the maximum and typical number. However the ascospores may multiply by budding before escaping from the ascus as well as after escaping. Ascospores measure  $3-4 \mu$  in diameter. When mature the asci open by an apical rupture and the spores escape.

**Overwintering.** — Previous to 1900 the opinion prevailed that the perennial mycelium in twigs was the chief method of overwintering. Sadebeck (12) stated that the mycelium occurred in the cortex and pith at the tip of one-year-old twigs. He claimed that this mycelium invaded the new leaves in the spring and accounted for most of the new infections. Beginning with the work of Pierce (10) published in 1900 evidence has accumulated which indicates that at least 95 per cent of the new infections must arise from spores rather than perennial mycelium. The proof for this lies largely in the fact that a dormant spray

will control the disease almost perfectly, which seems evidence enough that not much overwintering could result from perennial mycelium in the twigs. It seems probable, from the available evidence, that the spores produced on the leaves are wind disseminated, lodge on twigs and buds, and remain dormant in this position until the buds swell in the spring, at which time the spores germinate and infect the young leaves and blossoms as they emerge from the buds.

*Infection.* — Ecological factors undoubtedly play an important part in determining the amount of infection and the progress of the disease. The weight of evidence indicates that a cold, wet period coming after the buds have begun to open favors the development of leaf-curl. With this disease it seems that a period of cold accompanied by rain places the peach tree in a susceptible condition while at the same time affording conditions favorable for the growth of the leaf-curl fungus. Different species of fungi differ in their ability to attack under varying conditions. In case of some diseases the host seems to be more susceptible under conditions most favorable for host growth. In other cases a host is most susceptible under conditions unfavorable for the best growth of the host. (See Chapter V.) The latter seems to be the case with the peach. This is probably the reason why leaf-curl is apt to be more severe in regions near large bodies of water. Here the atmosphere is apt to be both more humid and cooler thus favoring the attacks of the leaf-curl fungus.

*Control.* — Numerous experiments in the United States during the last quarter of a century have demonstrated that peach leaf-curl can be controlled with comparative ease. One application of spray made at the right time is very effective. The spray can be applied at any time that is most convenient during the dormant season provided it is done before the buds swell. The last provision is essential and should be emphasized. Infection may take place as soon as the bud scales loosen, therefore the spray *must* be applied before that time. Any good sulfur or copper spray is effective. Bordeaux 6-6-50 is recommended although it is said that the 2-2-50 strength will control the disease. Lime-sulfur is also effective and if it is needed for scale or other dormant clean-up work it may be substituted for bordeaux. As a dormant spray it may be used at a strength of 1 to 8, if necessary, although 1 to 20 is strong enough to control the leaf-curl alone. Whatever spray is used two things are essential. These are: first, spray before the buds swell, and second, cover every bud thoroughly. Carelessness in either respect will result in failure.



## LABORATORY STUDY OF PEACH LEAF-CURL

## A. Symptoms.

1. *On leaves.* — Examine diseased leaves, fresh if available, otherwise both dried specimens and leaves preserved in liquid. If available observe young leaves showing the early stages as well as older stages in development, especially those showing the sporulating stage of the fungus. Note color and characteristic deformities resulting from the attacks of the fungus in both early and late stages. How do you account for the puckering and curling of the leaf? Note the comparative thickness of diseased and healthy leaves. How would you explain the thickening of the leaf? Examine sections through diseased leaves and study the cell structure. Compare with sections of normal leaf. Compare number, shape and size of cells. Are the palisade and spongy layers normal? Are there chloroplasts in the cells of the diseased leaf just as in those of the healthy leaf? Draw habit sketches of deformed leaves.

2. *On twigs.* — Examine diseased shoots. Does a diseased shoot elongate more or less than a healthy shoot? Compare the diameters of normal and diseased twigs of the same age. Do diseased shoots always grow as straight as healthy twigs regardless of length? Note that the dead, curled up leaves have a tendency to cling to the diseased twig long after it is dead. Draw diseased twigs.

3. *On blossoms and fruits.* — If diseased flowers and fruits are available note the symptoms. Are the flower parts deformed and thickened as the leaves are? Observe the diseased areas on the surface of fruits. Is there a thickening corresponding to that in leaves? Cut open the fruit. Are there any internal symptoms? If specimens of plum pockets, a plum disease caused by a closely related species of *Exoascus*, are available, compare with the diseased peaches and note differences and similarities. Draw diseased fruits.

## B. Morphology of the fungus.

1. Examine sections of diseased leaves under the microscope. Try to locate the mycelium within the leaf tissues. Find the layer of asci on the upper epidermis. Is the cuticle still in place? Count the spores in several asci. Draw a vertical strip through the leaf section showing the cell structure of the leaf and the relation of the fungus to the leaf tissues. Take especial care to show the layer of asci in place. Draw one or two individual asci enlarged.

2. Look up the question of overwintering. What are the two possible ways in which the leaf-curl fungus may be carried over from one year to the next? In view of the investigations of this disease during the last 25 or 30 years, which method of overwintering seems to be the chief one? When, where and under what conditions does infection occur?

## C. Field observations.

If season and other conditions permit, a very profitable field study may be made in the peach orchard. Any or all of the following items may be observed, depending on the time of year and other conditions. (a) Deformed and dead shoots killed the previous season. (b) Dead leaves killed last season and still clinging to the tree. These will usually be on the dead twigs mentioned above. (c) First signs of infection on young leaves soon after they burst from the bud. (d) Watch development of diseased leaves to maturity (requires repeated trips). (e) Observe results of control measures if any are being practiced in the neighborhood. (f) Estimate percentages of diseased leaves on sprayed and unsprayed trees. (g) Look for evidences of blossom and fruit



infection. (h) Try to estimate reduction in set of fruit and total loss due to the disease.

#### D. Notes.

Write complete notes covering all points of interest on this disease. Give particular attention to symptoms, life cycle and control.

#### REVIEW QUESTIONS

1. What is the original home of the peach leaf-curl fungus?
2. What is the present known distribution of the disease?
3. What is the nature of the damage caused by this fungus? What are the pathological effects on the host?
4. Name some other closely related species of *Exoascus* and their hosts.
5. Look up the work of Pierce (10) and find out what he contributed to our knowledge of this disease.
6. What did Atkinson (5) contribute to our knowledge of the species of *Exoascus*?
7. Describe the morphology of *Exoascus deformans*.
8. What are the ecological conditions conducive to severe outbreaks of peach leaf-curl?
9. What features of the life history of this fungus are most closely related to its successful control? State the seasonal limits within which spraying for leaf-curl must be done in order to be effective. Are these time limits calendar dates? Why?
10. In the usual spray practice now recommended for this disease, is the spray put on before or after the spores are disseminated? How does this compare with the practice for the control of late-blight of potato? How do you account for the fact that this spray practice controls leaf-curl in spite of the fact that spores are already on the buds when the spray is applied?

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### Witches'-Broom of Cherry

Caused by *Exoascus cerasi* (Fckl.) Sadeb.

The peach leaf-curl disease, previously discussed, is undoubtedly the most widespread and prevalent disease caused by species of *Exoascus*, but there are other diseases caused by different species of this genus which are well known on some of our stone-fruit trees. The witches'-broom of cherry and the pocket or bladder disease of plums are both caused by species of fungi belonging to the same genus as the peach leaf-curl fungus.

Cherry witches'-broom is characterized by the over-production of twigs on infected branches, giving rise to a bushy or broom-like growth which is well described by the common name in general use in America. In England other common names have been applied to the trouble, such as thunder-bushes and bull-boughs. The leaves growing on the brooms are usually curled very similarly to peach leaves affected by the curl disease. On cherry leaves the fruiting stage of the fungus produces a whitish or silvery coat, just as in peach leaf-curl, due to the presence of a layer of asci on the surface of the leaf. Some witches'-brooms are stiff and upright, while in other cases the growth may be more slender and pliable so that the broom becomes pendulous. Broomed branches produce few or no blossoms and the leaves appear earlier than on the healthy branches so that at blossoming time the brooms stand out in striking contrast to the rest of the tree (Fig. 65).

The mycelium of the fungus penetrates the tissues of the branch and lives there perennially without killing the twig. The fungus stimulates the production and growth of an excessive number of buds, thus giving rise to the supernumerary twigs found in the broom. Since the mycelium is perennial, when a twig is once infected the broom persists

indefinitely unless it is cut out. Although a crop of ascospores is produced every year on the diseased leaves of the broom, new infections do not seem to occur very frequently, so that as a rule brooms are not very abundant. For this reason the question of control has never

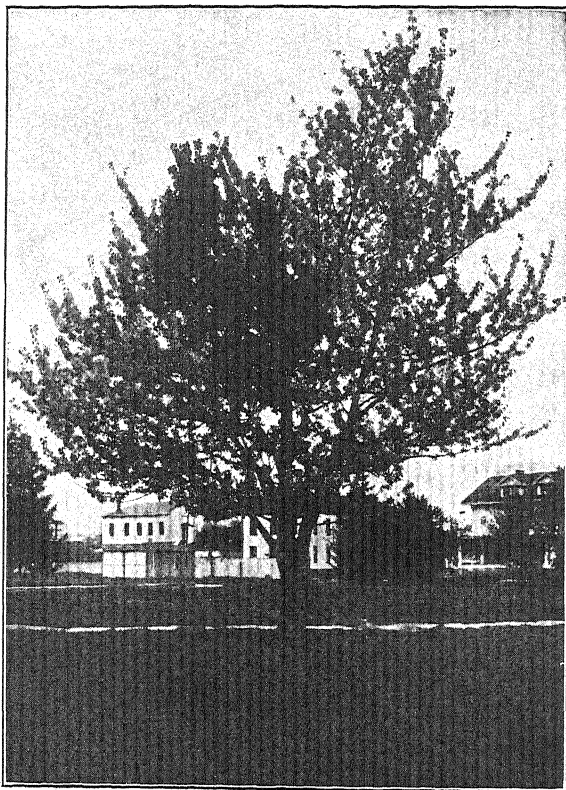


FIG. 65. — Witches'-broom in a cherry tree at blossoming time. The blossomless broom in full leaf shows in striking contrast against the white background of the remainder of the tree in full blossom.

received a great deal of attention. The usual control measure applied consists of cutting out the brooms. Since the disease does not assume epidemic proportions this expedient usually suffices.

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### Plum Pockets or Bladders

Caused by *Exoascus pruni* Fekl. and *E. communis* Sad.

Several species of *Exoascus* which attack plums have been described. The typical effect shows as a hollow, bladder-like condition of the green fruits. The fungus invades the young ovaries and inhibits the development of the pit, leaving a cavity surrounded by the spongy or leathery and more or less deformed outer fleshy part of the fruit. Diseased fruits may become much larger than normal ones. Affected plums are worthless. The fruiting stage of the fungus consists of a layer of asci over the surface of the fruit, resembling the fruiting layer on the surface of curled peach leaves.

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### American Brown-rot

Caused by *Sclerotinia fructicola* (Wint.) Rehm =  
*S. cinerea* (Bon.) Schröter forma *americana* Wormald =  
*S. americana* (Wormald) Norton and Ezekiel

This disease is widespread and destructive on stone fruits in North America and occasionally also attacks fruits of the pome type, particularly the apple.

**Historical.** — The brown-rot disease was frequently mentioned in American literature during the last quarter of the nineteenth century. In 1880 Peck (19) described brown-rot as one of the common fruit diseases. During that time, however, the causal fungus was considered to be identical with *Monilia fructigena*, a closely related species which had been known in Europe for almost a century. In 1851 a second species, *Monilia cinerea*, was described in Europe by Bonorden (3). The distinction between these two species of *Monilia* was not fully established until 1900 when Woronin (38) demonstrated that *Monilia fructigena* and *Monilia cinerea* are distinct species. In 1888 Woronin demonstrated that a sclerotinia occurring on a species of *Vaccinium* is the perfect stage of a *Monilia* occurring on that host. Following this discovery, it was assumed by Schröter (27) that *Monilia fructigena* and *Monilia cinerea* also are the imperfect stages of *Sclerotinia*, although no perfect stages of these fungi were known at that time. In 1905 Aderhold and Ruhland (1) discovered an ascospore stage for *Monilia fructigena*, demonstrated its relationship by means of cultures, and established the name *Sclerotinia fructigena* for that species. Meanwhile, in 1902, Norton (16) had discovered an apothecial stage for the common brown-rot fungus in America and demonstrated its relationship with the *Monilia* stage. Thinking our fungus to be the *Monilia fructigena* of Europe he named it *Sclerotinia fructigena*. Aderhold and Ruhland did not believe that the American fungus was *S. fructigena*, but considered it to be *Sclerotinia (Monilia) cinerea*. Later, American pathologists accepted this idea and for many years, until quite recently, the common American brown-rot fungus was called *Sclerotinia cinerea*. In 1919 Wormald (34), while making a study of certain strains of *S. cinerea* in England, compared the American fungus with his strains of *S. cinerea* and concluded that the American organism is a form of *S. cinerea* not occurring in Europe and named it *S. cinerea forma americana*. In 1924 Norton and Ezekiel (18) affirmed that the American form exhibits enough differences from the European *S. cinerea* to warrant creating a distinct species and they proposed that the common name, American brown-rot, be given to the disease and that the scientific name *S. americana* be used to designate the causal fungus.

For a long time the fact had been overlooked that as early as 1883 Winter (31) had published a description of a cup fungus, found on peach mummies in Pennsylvania, under the name *Ciboria fruticola*, which name was later changed by Saccardo to *Sclerotinia fruticola*. Of course, at that time Winter did not suspect any connection between this cup fungus and the brown-rot organism. In 1909 Pollock (20) called attention to Winter's description and stated that this cup fungus was

probably the perfect stage of our American brown-rot fungus. In 1924 and again in 1927 Roberts and Dunegan (24, 25) reviewed the history of this disease and finally concluded that our fungus is distinct from any European species and is undoubtedly identical with the species from Pennsylvania described by Winter, and should, therefore, be designated *Sclerotinia fructicola* (Wint.) Rehm.

During recent years another fungus of the *Monilia* type has been found in America, particularly in the Pacific Coast states, which causes a blossom-blight of fruit trees with but little decay of ripening fruit. This fungus is quite distinct from the common American brown-rot fungus and its identity has occasioned considerable doubt in the minds of plant pathologists. Now, however, this Pacific Coast *Monilia* is considered, by both American and European workers, to be closely related to if not identical with the true *Sclerotinia* (*Monilia*) *cinerea* of Europe. In this text, then, the term "American brown-rot" will be used to designate the widespread American disease found principally on fruits of the drupe type, and caused by a fungus which has been named successively *Monilia fructigena*, *Sclerotinia fructigena*, *Sclerotinia cinerea*, *Sclerotinia cinerea forma americana*, *Sclerotinia americana* and *Sclerotinia fructicola*.

**Geographical distribution.** — The American brown-rot disease as designated above is widespread throughout most of the fruit-growing sections of North America. Whether this species occurs in any foreign country is not at present known.

**Hosts.** — The stone fruits, including peaches, plums, cherries and apricots, are all attacked by *Sclerotinia fructicola*. To a lesser degree the pome fruits are also susceptible. The range of varietal susceptibility has not yet been definitely determined.

**Economic importance.** — Under favorable conditions of high temperature and humidity this disease is capable of causing enormous losses. The peach-growing states of the South suffer the heaviest losses from this disease. In 1900 the loss in Georgia was estimated at \$500,000 to \$700,000. In 1923 the same state lost 5 per cent of the peach crop, or 279,000 bu., according to the Plant Disease Reporter issued by the Plant Disease Survey of the U. S. Department of Agriculture (39). During the same year, South Carolina lost 30 per cent of the crop or 280,000 bu., and the total loss for the whole United States was estimated at 2.7 per cent, or 1,327,000 bu. In the prune-, cherry-, and peach-growing sections of the Pacific Coast states brown-rot frequently causes great loss.

**Symptoms.** — The disease develops on the fruits, blossoms and twigs and occasionally but not commonly on the leaves.

*On fruits.* — As the name signifies, the disease manifests itself as a brown rotten spot on the fruit, starting at a point and gradually enlarging until the whole fruit is rotted (Fig. 66). The diseased flesh is neither extremely soft and watery nor dry and firm but rather a medium soft rot with the skin over the rotten spot remaining more or less intact though turning brown like the flesh. After the fruit becomes thoroughly

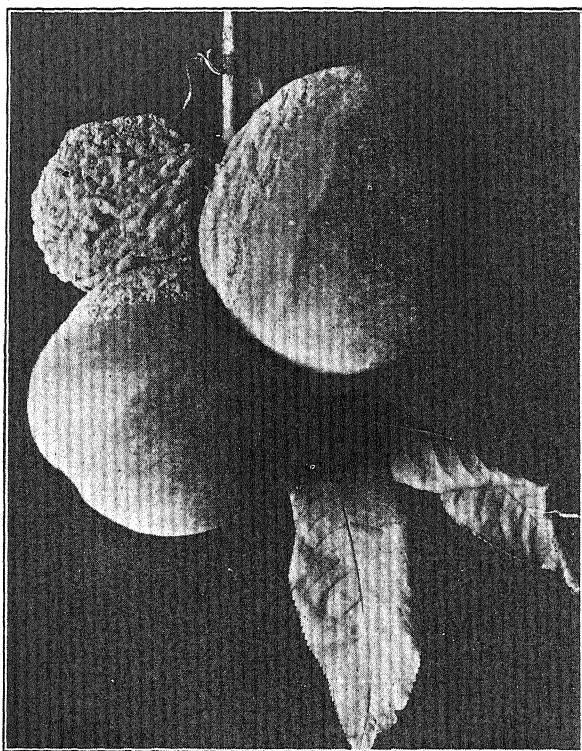


FIG. 66. — A ripening cluster of peaches attacked by brown-rot. The fungus spreads from one fruit to another when the fruits are in contact. (After Barss, Ore. Agr. Exp. Sta. Cir. 53.)

rotten the skin gradually turns black in color. If not destroyed the rotted fruit eventually shrivels into a mummy and the mycelium of the fungus which permeated the rotten flesh becomes a black, shrunken, dry, hard layer of resting fungous tissue, pseudoparenchymatous in nature, known as a sclerotium (Fig. 69). This sclerotium is very resistant to decay and if not destroyed may persist in the soil for several years. Early in the progress of the rot in a fruit the surface of the rotten area

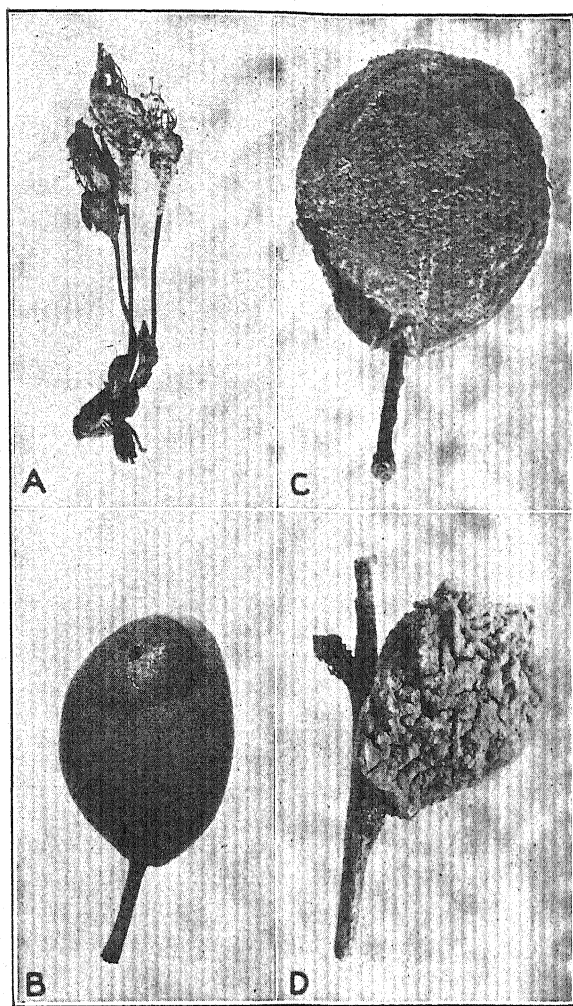


FIG. 67. — Several phases of the brown-rot disease. A, a cluster of blighted blossoms showing the ashy gray conidial growth of the fungus on the dead blossoms and the upper parts of the pedicels. B, a green prune artificially inoculated showing a small rotten spot around the point of inoculation. C, a mature prune completely rotted but not yet mummified, covered with conidial tufts. D, a completely mummified peach clinging to a twig, with its convoluted surface completely covered with a dense growth of conidiophores and conidia. (B and D from photographs in files of the Ore. Agr. Exp. Sta.)



may become more or less covered with small clumps of a grayish or ash-colored mold. This growth represents the asexual, conidial or imperfect fruiting stage of the brown-rot fungus (see under Life History), and is one of the chief signs or marks of identification of the disease.

*On blossoms.* — Blossom-blight is sometimes one of the chief sources of loss from the brown-rot fungus. Frequent showers or continuous rains at blossoming time sometimes induce a severe outbreak of blossom-blight. Infection may occur directly on the petals, in which case brown spots appear and these may spread until the whole petal turns brown and falls. Again, infection may occur on the stigma. In case of stigma infection the mycelium usually spreads down the style into the ovary

which of course results in fruit drop and a consequent reduction of stand. Not infrequently the loss in set of fruit from blossom-blight amounts to nearly 100 per cent.

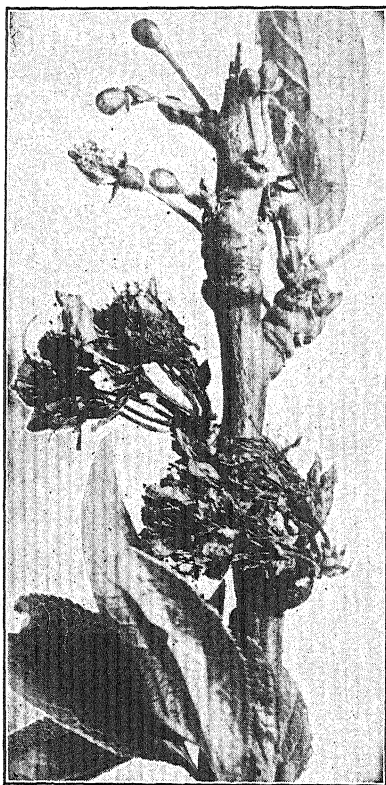


FIG. 68. — Brown-rot blossom and spur blight on prune. (After Barss, Ore. Agr. Exp. Sta. Cir. 53.)

*On twigs.* — The damage to twigs and branches resulting from attacks of the brown-rot fungus takes two forms, an outright blighting or killing of spurs and small twigs, and the formation of cankers on the larger twigs and small branches. In case of blossom-blight it frequently happens that the mycelium does not cease its spread when it reaches the ovary but continues into the fruit spur and kills it (Fig. 68). When spurs are blighted the mycelium sometimes continues spreading into the twig at the base of the spur and may either partially or entirely girdle it. In the latter case the twig is killed beyond the girdle, but if only partially girdled a canker is formed on the twig at the base of the spur. The dead spur

eventually drops off but the canker usually persists for a long time. In some cases a normal callus gradually heals the canker, but in other

cases a rough, gnarled canker is formed which may never heal properly but persists indefinitely as a disfiguring knot or swelling on the branch.

*On leaves.* — As a rule this disease is of no consequence on the leaves. Occasionally when leaves come in contact with a rotting fruit or cluster of fruits the mycelium may invade the leaf tissue and kill areas of the leaf or even whole leaves. Apparently spore infections do not take place at random on leaves as they do on fruits and blossoms.

**Morphology and life history of the fungus.** — The brown-rot fungus develops an extensive mycelium in the invaded tissues and reproduces by two spore forms. A sclerotium is concerned in the production of one of the spore forms.

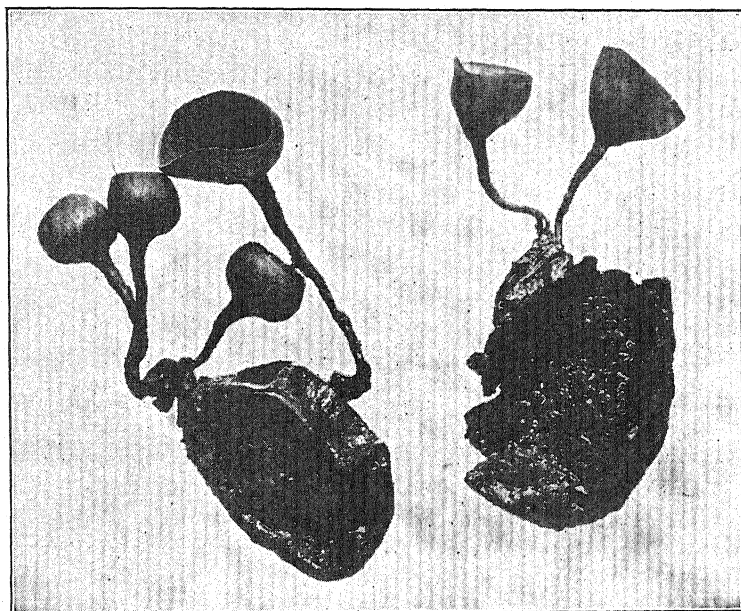


FIG. 69. — Apothecia of the brown-rot fungus arising from prune mummies. (After Barss, Ore. Agr. Exp. Sta. Cir. 53.)

*Conidial, imperfect or asexual stage.* — Usually before the rot has progressed very far in the fruit a moldy growth appears on the surface of the rotted area in the form of small, velvety clumps of fungous growth varying in size from minute specks up to one or two millimeters in diameter and ash-gray in color (Fig. 67, A, B, D). The average size of these cushions will usually approximate that of an ordinary pin head. Under extremely favorable conditions of humidity and temperature these conidial cushions may coalesce into a more or less continuous layer over

the surface of the rotted area. A microscopic examination will disclose that this moldy growth is composed of large numbers of branched conidiophores and that the branches consist of chains of conidia (Fig. 71). The conidiospores, when mature and separated from the conidiophore, are somewhat oval in shape with a papilla-like protrusion at one end which gives them a striking resemblance to a lemon in shape. They

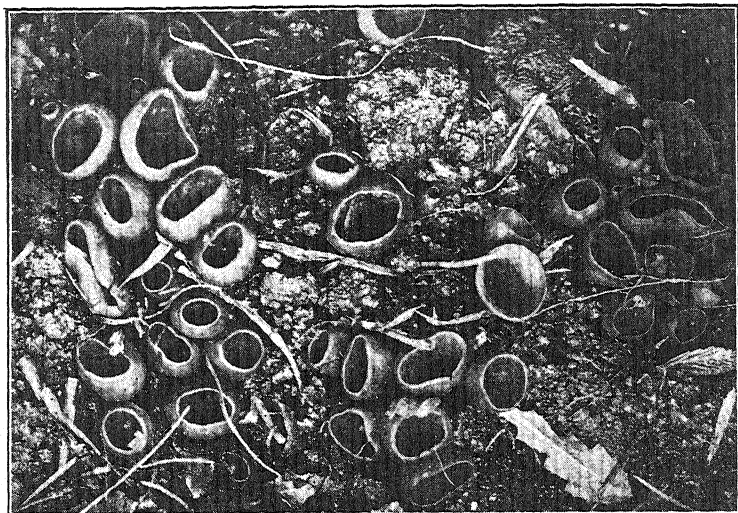


FIG. 70. — Brown-rot apothecia on the surface of the ground, viewed from above. The mummies from which they arise are buried in the ground. (After Barss, Ore. Agr. Exp. Sta. Cir. 53.)

average approximately  $15 \times 10 \mu$  in size. This conidial stage is produced not only on the fruits but also on blighted blossoms and spurs, and on twigs and cankers killed by the fungus.

*The perfect or ascogenous stage.* — This stage occurs only on fruits under certain conditions. As was described above, if a rotted fruit is allowed to lie on the ground undisturbed a sclerotium gradually forms from the mycelium as the flesh of the fruit decays, and eventually this wrinkled sclerotium comes to occupy the region immediately surrounding the stone or pit and thus replaces the flesh which has been destroyed. At about blossoming time the next spring, or more often the second spring following, and in some cases for several succeeding springs, there arise from this sclerotium, or mummy, cup-like structures known as apothecia (Fig. 69). These apothecia are fleshy brown cups varying in size from two or three millimeters in diameter up to approximately two centimeters in extreme cases. These cups rest on the ground but are

connected with the mummy by slender stalks which vary in length depending upon the depth to which the mummy is buried. Each cup is lined with a hymenial layer consisting of long slender asci each containing

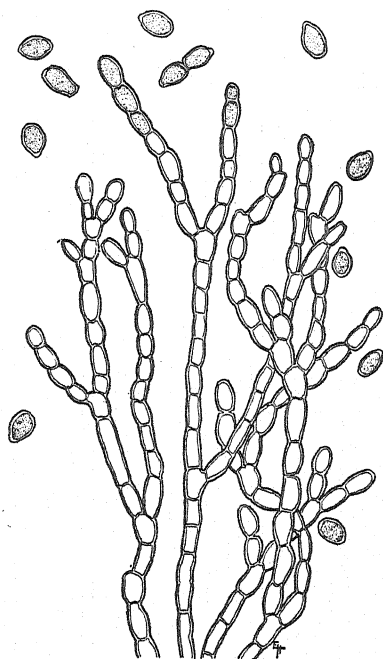


FIG. 71. — *Sclerotinia fructicola*. Conidiophores and conidia.

eight ascospores (Fig. 72). The asci are interspersed with sterile filaments called paraphyses. A single cup of average size may produce many thousands or perhaps millions of ascospores. The asci average about  $155 \times 9 \mu$ . The ascospores are somewhat elliptical in shape, single-celled, and measure approximately  $12 \times 6 \mu$  in size. When mature and under suitable weather conditions, the asci in the cup explode in large numbers, thus ejecting small clouds of spores into the air.



FIG. 72. — *Sclerotinia fructicola*. Section of an apothecium showing the asci and paraphyses of the hymenial layer which lines the inside of the spore-cup.

*Perpetuation.* — As has been noted there are two reproductive stages in the life history of the brown-rot fungus. Either of these spore forms may serve to perpetuate the organism.

The sclerotium from which the ascospore stage arises is capable of retaining dormant life for several years and producing apothecia during one or more succeeding spring seasons. Although brown-rot mummies ordinarily do not often produce apothecia until the second spring, there is evidence that, in some cases at least, apothecia are produced the first spring, that is, on mummies from fruit rotted the previous season. There is also evidence (21) that sclerotia ten years old are sometimes capable of producing apothecia.

Conidiospores may also initiate new infections in the spring. It is not likely that individual conidia survive the winter months but the mycelium of the fungus may live through the dormant months in the blighted blossom clusters, spurs, twigs and branch cankers, as well as in rotted fruits which remain hanging on the tree or even lying on the ground. On these lesions conidiospores are sometimes produced during open rainy weather in the winter or in early spring and thus blossom infections and early fruit infections may sometimes originate from conidia rather than from ascospores.

*Dissemination.* — Both spore forms of the brown-rot fungus are apparently largely wind disseminated. Insects may also play some part in disseminating the fungus.

*Infection.* — Infection may occur at any time from the blossoming period to maturity whenever extended periods of rainy weather occur. There are two periods in the development of the fruit, however, when excessive infection is likely to occur, especially if the weather conditions are favorable, namely at blossoming time and again as the fruit approaches maturity. Insects such as the curculio often cause large increases in brown-rot by puncturing the skin of the fruit and thus affording moist ports of entry for the fungus regardless of the weather. Rain also may cause more or less splitting of nearly mature fruits and this facilitates invasion by the brown-rot fungus.

*Control.* — The prevention or control of brown-rot may be discussed under two general heads, namely, field control, and the prevention of rot in transportation and on the market. For field control there are three chief means available: pruning, sanitation and spraying. Control in transit and on the market involves, first of all, field control; secondly, careful handling; and thirdly, control of environmental factors.

In controlling brown-rot from the production end it is necessary to consider both spore stages, since conidia may be produced on old rotted hanging fruits and on blighted spurs and twigs. All these should be

removed during pruning operations in so far as practicable. Proper sanitary measures require that all diseased parts be destroyed immediately upon removal.

To prevent the formation of the perfect or ascogenous stage, mummies should not be permitted to overwinter on the ground. If practical they should be destroyed in some manner. It has been shown that plowing mummies under to a depth of two or three or more inches will prevent the formation of apothecia. However if the land is plowed again the following year and the mummies brought to the surface again, apothecia may be produced from such mummies.

Since the pruning and sanitary measures mentioned, even if faithfully observed, have never been known to entirely eliminate brown-rot, it often becomes necessary to resort to spraying to save the crop. It is not possible to recommend a uniform spray program that will apply to all localities in all seasons. In some regions where blossom- and spur-blight are important factors it may be necessary to apply two or three sprays for the prevention of this type of loss. In other cases it may be necessary only to protect the fruit from decay as it approaches maturity. A full schedule for severe cases is recommended by the Oregon Experiment Station (2) as follows:

1. *Pre-blossom spray*. — Bordeaux 4-4-50 (or lime-sulfur 1 to 50) just before the blossoms open, when the petals are showing white, to protect against blossom-blight.
2. *Petal-fall spray*. — Bordeaux 4-4-50, lime-sulfur 1 to 50, self-boiled lime-sulfur 8-8-50, dry-mix sulfur and lime or atomic sulfur, etc. (see Chapter VII), when petals are gone. For blossom-blight.
3. *Shuck-fall spray*. — Self-boiled lime-sulfur, dry-mix sulfur and lime, etc., or sulfur dust. Protection for young fruit.
4. *Summer sprays*. — Same materials as in 3. Use whenever danger of brown-rot is evident, to protect developing fruit.
5. *Pre-harvest spray*. — To protect ripening fruit. Use same materials as in 3 and apply about a month before harvest.

Each grower will have to determine for himself how many of these sprays he can profitably apply. The answer must be based upon the conditions under which he works and his past experience. In some cases it may be necessary to protect only the maturing fruit. Possibly one application, number 5 above, may be all that is necessary. It all depends upon circumstances, and the observant grower will soon learn to judge as to how many applications will be necessary to save his crop. He must study the situation intelligently and act according to his best judgment, of course using all the reliable advice he can get from any source.

*Transportation and market problems.* — Stone fruits are largely marketed in fresh condition when nearly or fully mature. In this condition they are very susceptible to rots unless extreme care is used in handling them. If brown-rot is present in the field before harvest many fruits are likely to carry incipient infection when harvested. Even when incipient infection does not exist spores may be lodged on the surface of fruits ready to cause infection under suitable conditions. Field control, then, is necessary in order to avoid certain danger after harvest. Careful handling to avoid wounds is also necessary, since bruises and broken skin facilitate infection by brown-rot spores. High temperatures favor spore germination, infection, and also rapid development of rot after infection (26). New infections rarely occur at temperatures below 50° F. The progress of rot already present is checked somewhat at this temperature but it requires much lower temperatures to stop it entirely. Loading before cooling and delay in transit together with faulty icing of the car may cause great loss.

*Summary.* — The control measures for brown-rot may be summarized briefly as follows:

1. Prune out and burn hanging mummies, blighted spurs and twigs.
2. Destroy or plow under all rotted fruit or mummies. Hogs may be turned in to eat them.
3. Follow a suitable spraying or dusting program.
4. Handle fruit carefully in harvesting and marketing.
5. Maintain low temperatures and good ventilation in storage or in transit.

#### LABORATORY STUDY OF BROWN-ROT

##### A. Symptoms and signs.

1. *On the fruits.* — Examine specimens of peach, cherry, plum or any other fruits attacked by this fungus. Note color, consistency and extent of the rot in the flesh, and also the tufts of conidiophores and conidia appearing on the surface of the rotted fruits. Make a drawing to represent these features. Now examine old mummified fruits which have lain on the ground for a year or two (about 18 months). Note the condition of the fleshy part of the fruit. What has happened to it? Observe also the cup-like fruiting bodies attached. Draw a mummy with attached cup.

2. *On twigs, spurs and blossoms.* — Examine twigs, spurs and blossoms which have been attacked by the brown-rot fungus. Note cankers on the twigs, and also observe the spurs that have been killed and the blossoms which have been blighted by the fungus. Look for conidial tufts on the blighted spurs or other killed parts. Make such sketches as will best show the effect of the disease on these parts of the tree.

##### B. Morphology and life history of the causal organism.

1. *The vegetative mycelium.* — Tease out a small fragment of the rotten pulp of a freshly decayed fruit in a drop of water, mount and observe under the microscope. Find hyphae of the fungus among the cells of the fruit and draw. If pure cultures

are available it may be more convenient to make mounts from these for study of the vegetative mycelium.

2. *The sclerotium.* — Examine a completely mummified fruit that has lain on the ground for many months. Cut into the black crust surrounding the pit or stone and note the nature of this structure. Examine this material microscopically. Use thin sections or tease out a small fragment of it in a drop of water on a slide and mount. What is the microscopic structure of the sclerotium? Of what does it consist, fungous or host tissue? What is its function?

3. *The conidial or imperfect stage.* — Mount conidiophores and conidia for microscopic examination. Note the shape and manner of branching of the conidiophores, and also the chains of conidia attached. **Draw.** What part does this stage play in the life cycle of the fungus?

4. *The ascigerous or perfect stage.* — Examine longitudinal sections of the cup (called apothecium). Note the main body of the cup and the lining layer (hymenium) composed of asci and sterile cells (paraphyses). Make a **diagram** of the whole section showing the different layers, especially the hymenium. Now make a large scale drawing of a narrow section of the hymenium showing a few asci and paraphyses much enlarged. If the prepared section is not suitable for this drawing, take a small fragment from the hymenium of a fresh cup or a preserved one, tease out in water, and make your own temporary mount. Find the asci and draw. How many ascospores in each ascus? At what season of the year do apothecia appear? From what do they arise? What rôle does this stage play in the life cycle of the fungus?

5. *Perpetuation, dissemination and infection.* — After working out the morphology of the fungus and reading the discussion in the text you should be able to discuss these important phases of the life cycle. How does the fungus hibernate? How is it disseminated in the various seasons? Where, when and under what conditions does infection occur? What are the conditions favorable for blossom and spur blight, and for fruit rot?

#### C. Cultural studies.

If facilities and time are available it may be desirable to make pure cultures and inoculations with this fungus.

1. Review Chapter V if you do not know how to make pure cultures of the fungus. Describe two ways in which artificial cultures may be secured.

2. After the fungus has been secured in pure culture, various inoculations may be made. The simplest and easiest inoculation to make is to bring some susceptible fruits into the laboratory and keep them in covered dishes. Following directions in Chapter V, describe the method or methods by which the inoculations may be accomplished. Carry out all the steps in Koch's postulate. If desired the more difficult field inoculations to produce blossom and twig blight may be performed. If it is desired to secure the perfect stage from the rotted fruits what procedure must be followed?

#### D. Field study.

If season and weather permit, make a field study of as many of the following features as are available.

1. Rotted fruits showing conidia, either hanging on the tree or lying on the ground.  
2. Twig cankers, blighted spurs and blossoms. Look for conidial tufts on the blighted parts. Try to determine the point at which infection entered in any particular case.



3. The sclerotia producing the apothecial stage. At what season of the year can cups be found, if at all?

#### E. Notes.

Write in detail everything you have learned about brown-rot and its causal fungus in any of your studies, in laboratory, in the field, and from your reading.

#### REVIEW QUESTIONS

1. What kinds of fruits are attacked by the brown-rot fungus?
2. Describe the symptoms on the different parts of the host such as fruit, blossoms and twigs.
3. Through what does spur infection usually take place?
4. What is the rôle of the sclerotium of the mummified fruit in the life cycle of the brown-rot fungus?
5. During what known age limits will a sclerotium produce apothecia?
6. What are the environmental factors necessary for the production of apothecia?
7. Does any correlation exist between the maturing of ascospores and the blossoming period of the host?
8. Which spore form is responsible for the primary infections? For secondary infections?
9. What weather conditions are conducive to an outbreak of blossom blight?
10. At what stage of development of the fruit is an outbreak of fruit rot most likely to occur? What environmental conditions favor such an outbreak?
11. How deep must a mummy be buried in the soil to prevent the production of apothecia?
12. What control measures are applicable as far as the ascospore stage is concerned?
13. What control measures must be used to combat the secondary spread, by means of conidia, to the maturing fruit?
14. Discuss the problem of controlling brown-rot on fruit marketed in a fresh condition. (See Chapter XIII.)
15. What part, if any, do cankers play in the overwintering of the fungus?

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### Northwestern Apple-tree Anthracnose

Caused by *Neofabraea malicorticis* (Cordley) Jackson

In the Pacific Northwest, especially west of the Cascade Mountains, this is one of the major diseases affecting apple trees. It is particularly serious as a bark canker but also occurs as a fruit-rot under the proper weather conditions. Previous to 1900 the disease was known locally under various names, some of which are "black spot," "dead spot" and "canker." Since 1900 the name "apple-tree anthracnose" has come into general use. The name "northwestern anthracnose" has more recently been suggested for this disease in view of the fact that it is confined to that part of the country and also because the name "anthracnose" was at one time applied to bitter-rot, an apple disease of the eastern half of the United States.

**History and distribution.** — According to Cordley (2) the apple-tree anthracnose had attracted considerable attention as a canker disease in the northwest for several years prior to 1900. In compliance with requests for information on the nature and control of the disease, N. B. Pierce was detailed by the U. S. Department of Agriculture to investigate the trouble (7). During 1894 and 1895 he made a study of the disease in Oregon and Washington, but apparently no official report of his findings was ever published. By 1899 the desire on the part of growers for information about the disease led to an investigation of the trouble by the Oregon Experiment Station. A. B. Cordley undertook the investigation and discovered the true cause of the malady to be a fungus. He published the first official account of the disease in 1900 (2). He cultured the organism and by means of inoculations on apple branches established its pathogenicity. The fungus was named *Gloeosporium*

*malicorticis* by him, and the popular name "apple-tree anthracnose" was proposed for the disease. In 1904 Lawrence (8) of the Western Washington Experiment Station confirmed Cordley's findings and contributed considerable additional information on the disease, notably the fact that the fungus causes a fruit rot as well as a bark canker. In 1906 Cate (1) began extensive field observations as well as cultural work in which he acquired information on the susceptibility of apple varieties and the host range of the fungus. In 1913 Jackson (7) published the results of his investigations begun in 1909. His chief contribution was the discovery of the perfect stage of the causal fungus, a discomycete which he named *Neofabraea malicorticis* (Cordley) Jackson.

The disease occurs throughout Oregon, Washington and British Columbia. It also has been reported from Northern California. It is not known to occur in any other part of the United States. In its most serious form it is confined to those parts of Oregon, Washington and British Columbia lying west of the Cascade Mountains. The fungus seems to require the mild and rainy winter season of this part of the country for its development in a serious form.

**Hosts and varietal susceptibility.** — This disease is confined largely to the apple, though it is frequently found on the pear. What appears to be the same fungus has been reported several times on the quince, especially as a fruit rot, but further careful work should be done to determine definitely whether this fungus attacking the quince is identical with the apple organism. Lawrence (8) reported finding similar cankers on cherry, plum and prune but gave no definite proof that they were identical. Both Lawrence and Cate (1) inoculated cherry, plum and prune branches with the apple anthracnose fungus and secured small cankers, but no spores were produced on these cankers. Pierce intimated (7) that he had found a native host for the organism but since he did not publish it no information on that point is available. The sources of information at present available indicate that this particular disease occurs naturally on the apple and pear and probably on the quince, but probably does not attack the stone fruits in nature. The writer has never seen any symptoms on the latter type of fruits which could certainly be attributed to the apple anthracnose fungus.

Few, if any, varieties of apples are immune to this fungus but there is considerable variation in the susceptibility of different varieties. From his extensive field survey of the disease Cate (1) concluded that the Baldwin, Spitzenberg and Jonathan are the most susceptible varieties, with Ben Davis, Northern Spy, Winesap and Blacktwig showing the most resistance, and Newtown, Greening, Gravenstein and most of the summer varieties ranking intermediate in susceptibility. The



FIG. 73. — Anthracnose canker on apple branch. Note pustules in the bark of the canker. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest and Hort. Rept. 1911-12.)

different varieties are not constant in their degree of susceptibility, however, and under certain conditions varieties which ordinarily seem resistant may become very susceptible.

**Symptoms and signs.** — The symptoms consist of the branch cankers and rotten spots on the fruits. The first indication of the disease on the branches consists of a small, round, discolored spot on the bark. This area is darker in color than the surrounding healthy bark and is more evident when the bark is wet, being scarcely noticeable at first on the dry bark. When the spots are one-half to one inch in diameter a slight zonation is sometimes visible, showing as a lighter spot in the center with one or more light zones occurring at intervals between the center and the margin of the canker. In many cases, however, this zonation is not evident. As the canker enlarges it usually elongates parallel to the axis of the branch, becoming oval or elliptic in shape. Mature cankers are of any size from approximately an inch up to several inches, or in exceptional cases, a foot or more in length. Typical cankers usually range from two or three to five or six inches in length. In some cases cankers girdle the branch but more often they are confined to one side of the branch (Fig. 73). The young cankers first

appear a few weeks after the autumn rains begin so that usually by mid-

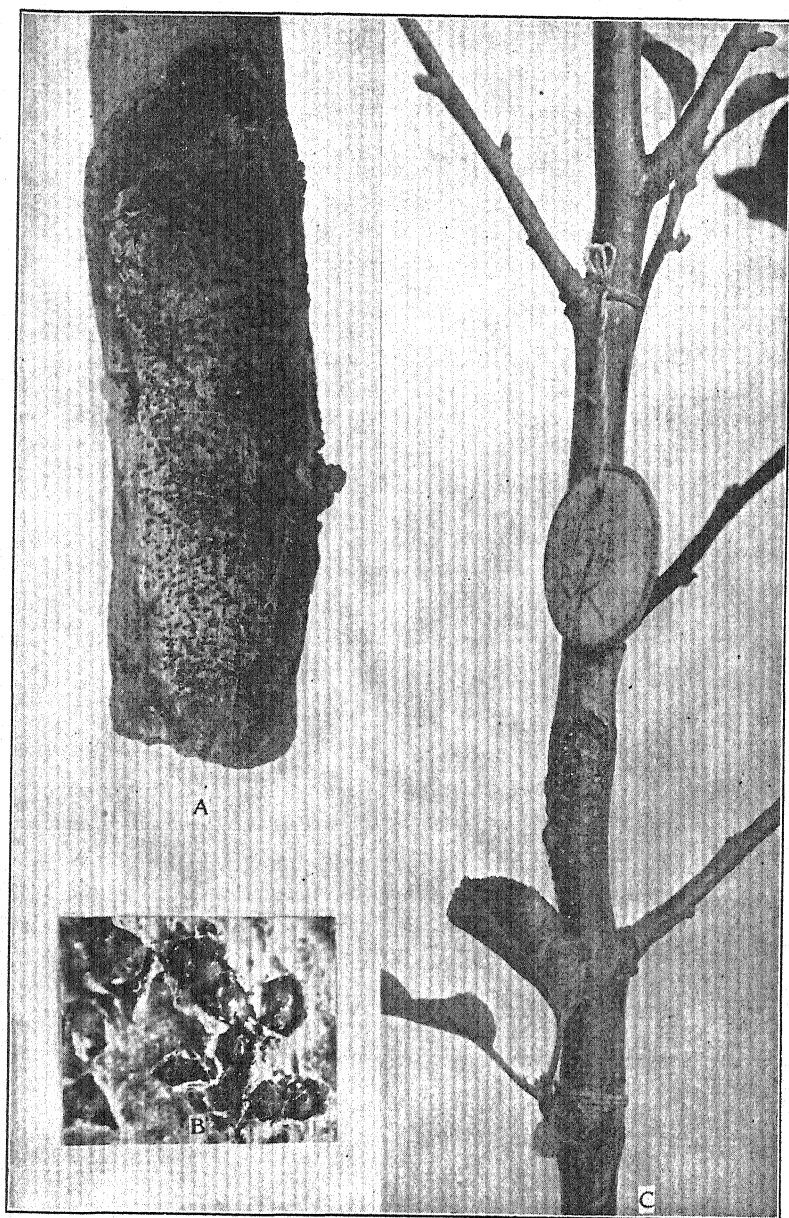


FIG. 74. — A, canker two years after infection showing apothecia of the perfect stage occupying the position of the conidial pustules of the preceding year. B, apothecia in bark, enlarged five diameters. C, young apple tree showing a canker with the conidial fruiting stage of the fungus, resulting from inoculation with a culture from the ascospore stage. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest and Hort. Rept. 1911-12.)

winter many small cankers are in evidence. The fungus spreads in the bark slowly during the winter but as spring approaches the cankers enlarge rapidly reaching their maximum size soon after the cambium becomes active in the spring. During the winter the cankered area is smooth and neither sunken nor elevated but when the enlargement of the canker ceases the dead area becomes sunken due to the drying of the dead bark and the formation of a callus around the canker. Soon a crack appears between the dead and the live bark and the canker thus

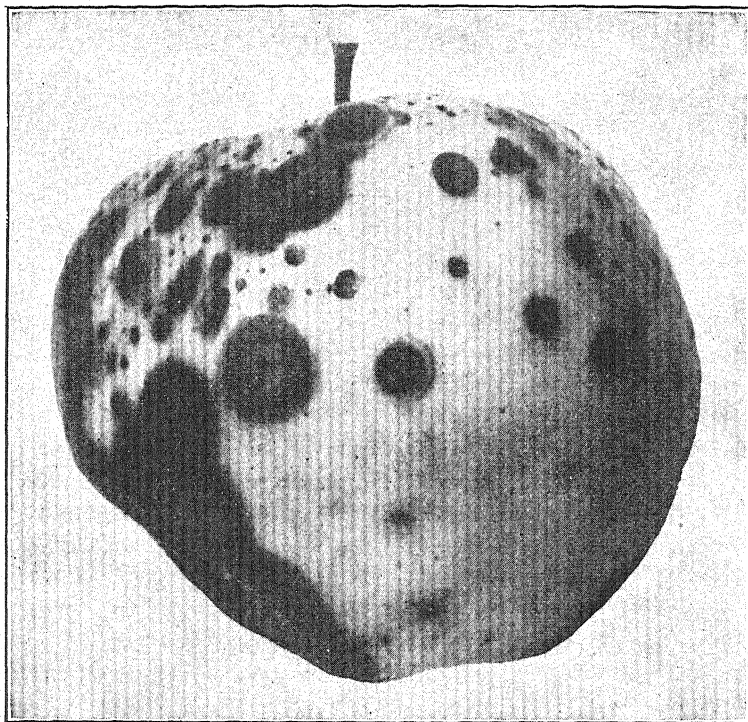


FIG. 75. — Anthracnose rot on apple fruit. (Photograph by Barss, Ore. Agr. Exp. Sta.)

becomes clearly marked in outline. As the season progresses the fruiting pustules of the fungus begin to appear. A few acervuli sometimes develop quite early in the spring but usually they are rather scarce on the cankers until autumn approaches. Sometimes a considerable number of pustules appear in June and later through the summer but with the approach of the rainy season they break out in abundance all over the cankers. The acervuli first appear as tiny raised papillae.



Later the epidermis over these elevations is ruptured and the waxy, cream-colored cushions of conidiophores and conidia push through. When the fruiting of the fungus is at its peak the canker is thickly dotted with these spore cushions which average one-half to one millimeter in diameter and arise from a crater-like cleft in the epidermis of the bark. Later, when the sporulating stage is past, these irregular craters with the dark-colored openings remain scattered densely over the dead surface as one of the chief signs by which the identity of the canker can be determined in the field (Figs. 73, 74 A).

On the fruit the disease first appears as small circular rotten spots. The number of these spots varies from one to many and as they enlarge two or more spots may coalesce to form larger rotten areas. Before the spot becomes very large it commonly acquires a more or less zonate appearance resembling in this respect the condition sometimes shown by the cankers (Fig. 75). Sooner or later acervuli may appear on the surface of the fruit lesion as well as on the cankers. It is not unusual for the fruiting pustules of the fungus to occur in concentric zones over the surface of the rotted area although this is not always the case. The surface color of the lesion is brown with sometimes the lighter zones mentioned above. The color of the rotted flesh is also brown and its texture is rather firm but not dry nor tough. The rotted portion assumes a cone shape with the base of the cone at the surface of the fruit and its apex pointing toward the center of the fruit, as is usual with fungous rots in fruits. The rotten spots enlarge slowly since the fungus does not make a rapid growth.

**Economic importance.** — The chief injury caused by this disease is due to the canker phase although under particularly favorable conditions the fruit-rot may be of considerable importance. In the apple-growing sections where anthracnose prevails, this disease is usually considered to be about equal to apple-scab in seriousness. The principal injury from scab, however, is inflicted directly upon the fruit and is therefore strictly a matter of seasonal importance while the chief damage from anthracnose consists of the more permanent damage resulting from the cankers and is cumulative on the tree if allowed to go unchecked. Thus, even though the direct loss from scab in a bad year might be greater than from anthracnose, in the long run anthracnose may cause more permanent injury in an orchard and thus permanently reduce the earning power of the orchard. In neglected orchards the cankers may become so numerous as to cover most of the branches, rendering them very rough and irregular in shape and even completely girdling many of them. Even where girdling does not occur to any extent the area of dead bark resulting in the aggregate from a large



number of small cankers may be such as to materially weaken the tree and thus greatly reduce the quantity as well as the quality of the fruit produced. During seasons when enough rain falls before harvest to bring about infection, if the fruit is not protected by a covering of spray, serious loss from rot may occur in storage, especially in common storage where the temperature is not low enough to prevent the growth of the fungus.

**Morphology and life cycle of the fungus.** — The fungus has two spore stages, the conidial or imperfect stage, and the perfect or ascogenous stage. The former is the common reproductive spore form while the

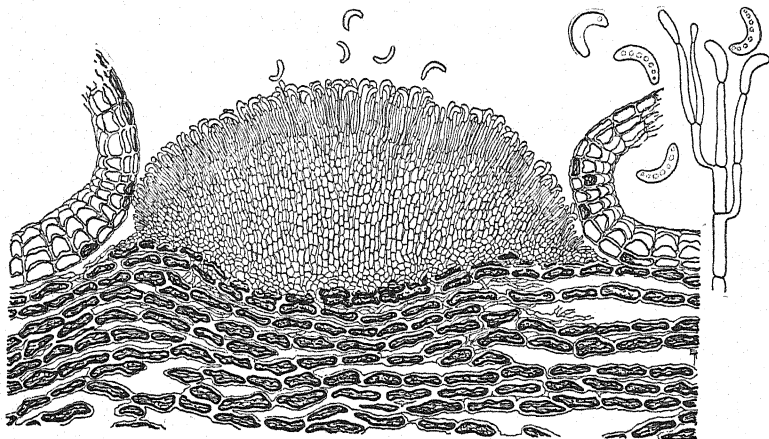


FIG. 76. — Section of mature acervulus of the apple anthracnose fungus. At right, conidiophore and conidia enlarged.

perfect stage occurs less frequently. The conidial pustules (acervuli), as indicated above, mature mostly in the fall about a year after infection occurs. A section through an acervulus (Fig. 76) shows a cushion of fungus tissue over the surface of which is a dense compact growth of conidiophores bearing conidia. The conidiophores are slender and considerably branched (Fig. 76), and the conidia are borne singly on the apex of each branch. The conidiospores are curved, hyaline, single-celled, and somewhat guttulate (Fig. 76). They average about  $6 \times 24 \mu$  in size. The degree of curvature varies from almost straight to the typical allantoid, or sausage shape.

The perfect stage consists of an apothecium which develops on two-year-old cankers. This ascocarp arises in the old conidial stroma under the old cushion of conidiophores and pushes up through this conidial layer which is pushed aside and sloughs off (Fig. 77). The apothecia

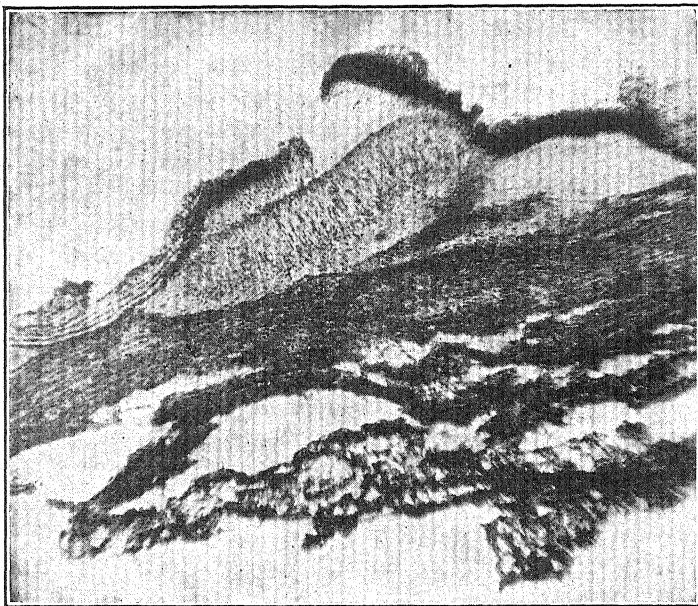


FIG. 77. — Photomicrograph showing the development of the apothecium of *Neofabraea malicorticis* in the old acervulus of the conidial stage. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest and Hort. Rept. 1911-12.)

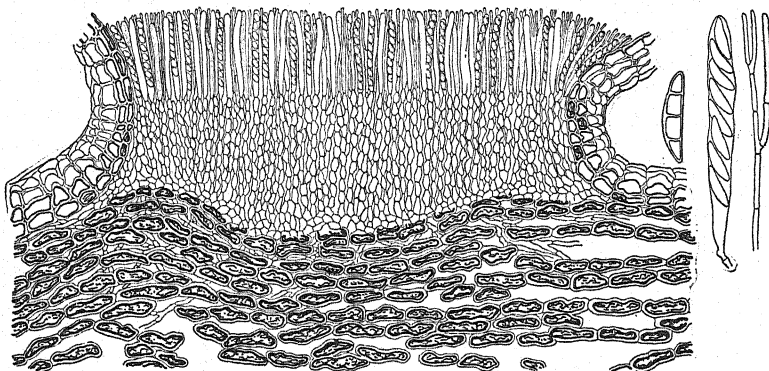


FIG. 78. — Section of mature apothecium of *Neofabraea malicorticis*. At right, ascospore, ascus and paraphysis enlarged. Ascospore becomes four-celled at maturity.

are rather waxy in consistency, light-brown in color, and measure one-half to one millimeter in diameter. The asci (Fig. 78) are club-shaped and measure 90 to 100 by 10.5 to 13  $\mu$ . The ascospores are hyaline,

elliptical, slightly flattened on one side, one-celled, becoming two- to four-celled upon germination, and measure 16 to 19 by 5 to 7  $\mu$ .

The fungus is perpetuated as vegetative mycelium in the bark of cankers, persisting for two or three or more years. Conidia are produced in greatest abundance on one-year-old cankers but may be produced on the dead bark of cankers two or more years old. Ascospores are produced on two-year-old cankers. It should be noted that both conidia and ascospores, when the latter occur at all, are mature and disseminated mostly immediately after the rainy season begins, usually in October or November. Dissemination is effected principally by rain and wind so far as known. Infection occurs during several weeks following the opening of the rainy season. Infection apparently takes place in the absence of wounds since most cankers occur on the smooth bark between nodes where there is no indication of wounds. Possibly lenticels offer a point of access but this question has not been investigated thoroughly. This fungus is apparently a low temperature organism. Cordley (2) states that the spores germinated readily at 72° F. but at 84° F. no germination occurs. The minimum temperature for spore germination is not known but evidently it is quite low since infection occurs in rainy weather during November and December when the mean temperature in the Willamette valley is about 40° to 45° F. In the light of these temperature data it is doubtful if extensive infection would occur in mid-summer even if spores were present and sufficient precipitation occurred. In addition to these limitations it was noted above that the increase in size of cankers is inhibited as soon as the cambium becomes active in the spring. Even if infection should occur during the growing season, the spread of the fungus in the bark would no doubt be inhibited by the formation of a layer of wound cork immediately surrounding the point of infection and thus no noticeable canker would appear. At any rate infection and the growth of cankers are apparently confined to the dormant period of the tree whatever may be the factors concerned in limiting the activity of the fungus to this season.

**Control.** — The measures which can be applied in controlling this disease are just two: (a) eradication of cankers and (b) the use of protective fungicides. The former measure is of practical use only on small trees and where relatively few cankers occur. Small canker-bearing twigs can be cut out completely and cankers occurring on larger limbs can be removed by shaving off the dead bark. But by far the most practical method available in the majority of cases is the use of fungicides. The most effective spray material thus far found is bordeaux mixture. It has been known for a long time that a thorough spraying

with bordeaux, covering every bit of bark on the tree, will almost completely prevent any new infections provided the spray is put on before the rainy season begins. This provision is vital and must be followed or failure will ensue. The most important problem now before growers and pathologists is the question as to the most effective as well as most appropriate time at which the spray can be applied. Must the spraying be done immediately preceding the beginning of the fall rains or can it be done some weeks or even months before the opening of the rainy season? This is a vital question since it is not desirable to spray just before harvest on account of the objectionable deposit left on the fruit. In former years it was customary to advise spraying as soon as the fruit was harvested. In practice it was found that in some seasons the rains came so early that it was impossible to get the spray on in time to be effective, if not applied until after harvest. Experiments were then begun to determine how long a good coating of bordeaux would stick on the bark and prevent anthracnose infections when the rainy season began. It has been shown that a properly applied bordeaux spray put on at any time during the summer or even in the early spring will remain on the tree and be effective against anthracnose the following autumn. The only question remaining, then, is one of convenience. Probably the most convenient and economical way to handle the situation is to apply the bordeaux as one of the scab sprays or with one of the codling moth applications. To avoid the objectionable deposit on the fruit this should be done not later than the July spray and probably one of the earlier applications would be better.

The above discussion of control measures applies to the canker phase of the disease. It is self-evident that if the disease is controlled in this respect there will be no fruit-rot problem. However, if an orchard is severely infested with the disease there is likely to be some fruit-rot in those seasons when considerable rain falls before harvest. The logical inference, of course, is that a vigorous control program applied to the canker phase will eliminate all danger from fruit-rot. The summer application to prevent canker will no doubt protect the fruit against rot to a certain extent when spores are present and rains fall before harvest. Where opportunity for fruit infection occurs before harvest about the only practical recourse is cold storage, with temperatures low enough to hold the development of the fungus in check.

#### LABORATORY STUDY OF APPLE ANTHRACNOSE

A. Symptoms. — The symptoms of this disease appear in the form of both branch cankers and a rot of the fruits.

1. *On branches.* — Examine branches showing lesions in different stages of development. Specimens collected in late winter or early spring will show the incipient

cankers. Note the shape, size and color of these young cankers. Is the surface of the dead bark smooth or rough, raised or sunken? Examine one-year-old cankers. How do they differ in appearance from the younger cankers? Observe especially the margin of the canker and the surface of the dead bark. What is the nature of the markings over the surface of the dead area? Examine two-year-old cankers. How do they differ from the one-year-old or younger cankers? Make drawings to illustrate all canker symptoms.

2. *On fruits.* — Examine fruits rotted by the anthracnose fungus. Note the color and texture of the rotted flesh and also the tendency toward color zonation on the skin of the rotten spot. Are there any fruiting bodies of the fungus present? Note the size, color, structure, and distribution of these pustules. Draw.

B. *The causal fungus.* — If material is available study both the conidial and perfect stages of the fungus.

1. *Conidial stage.* — The acervuli occur on the one-year-old cankers and on the rotten fruit. Examine pustules with dissecting microscope or hand lens. Note the manner in which the outer bark is ruptured and the conidial cushion protruding from the pustule. This will show better on bark that has been kept moist for a while before the study begins. Examine prepared sections of acervuli with the microscope. Note the conidiophores and conidia. What is the characteristic shape of the conidiospores? Draw sectional view of one acervulus in detail. Draw individual conidiospores much enlarged.

2. *Perfect Stage.* — Where should one expect to find the perfect stage? (See text.) Does it occur as abundantly as the conidial stage? Examine with a lens the apothecia in place on the canker. Compare in general appearance with the acervuli. Examine sections of apothecia under the microscope and draw a sectional view of one apothecium. Draw a single ascus and contained ascospores much enlarged.

3. *Perpetuation, dissemination and infection.* — Look up the life history of this fungus in the text. In what form and where is it perpetuated from year to year? At what season of the year is the main crop of spores matured and disseminated? Does this apply to both conidiospores and ascospores? When, where and under what circumstances does infection occur? Discuss the preceding question for both canker and fruit rot.

4. *Cultures.* — This fungus is readily isolated and grown in pure culture. Isolations can be made easily from both cankers and rotted fruits by the tissue culture method described in Chapter IV. If facilities and time are available, make cultures and try some inoculation experiments on both branches and fruits.

C. *Notes.* — Write a complete account of this disease following the outline on page 152. Give particular attention to the life history, especially to the time of infection as related to control measures.

#### REVIEW QUESTIONS

1. What is the known geographic distribution of the north western apple-tree anthracnose disease?
2. State the characters which serve to distinguish the cankers of this disease from all other canker diseases occurring on apple trees.
3. Describe the symptoms of the fruit-rot caused by this fungus.
4. Describe the two fruiting stages of the anthracnose fungus.
5. How is the fungus perpetuated from year to year?
6. During what season of the year is the fungus most active?

7. At what season of the year does each type of spore produced by the anthracnose fungus mature? In each case how long is this after infection occurs?

8. Is there any correlation between the time of spore maturity and the season at which infection occurs?

9. Based upon the nature of the disease and the life history of the fungus, what two control measures are applicable to this disease?

10. What feature of the life history determines the time for applying the spray to prevent canker infection?

11. What practical considerations in growing the apple crop enter into the spray program for this disease?

12. Discuss the necessity for and the application of control measures for the fruit-rot phase of this disease.

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### The Powdery Mildews

Caused by fungi of the family Erysiphaceae

The term "powdery mildews" is applied to a very large group of diseases caused by fungi belonging to the family Erysiphaceae. The powdery mildews as a whole are widely distributed over the world and their range of hosts includes a very large number of families and species of plants. Among the plants which are subject to attack by one species or another of the powdery mildew fungi are the common fruits, such as apple, peach, cherry, grape, gooseberry, strawberry and the bramble fruits; many shrubs and herbaceous plants including roses, lilacs, peas, cucurbits and a large number of weeds; broad-leaved forest trees such as the oak, maple, dogwood, willow and alder; and many members of the grass family including the cereals and numerous wild grasses. The economic importance of the powdery mildews is very great in the

aggregate. In some cases the damage done by them is very slight and may be entirely negligible, while in other cases severe attacks may result in serious or even total loss of certain crops. On some varieties of grapes, gooseberries and roses, for example, serious infestation by powdery mildews is common and disastrous. In many cases the injury is incidental to the lowering of photosynthetic efficiency of the mildewed foliage and while no crop failure may result there undoubtedly is more or less reduction in yield which is difficult to estimate accurately.

**Symptoms and signs.**—The powdery mildews are characterized chiefly by the presence of the causal fungus, in both its vegetative and

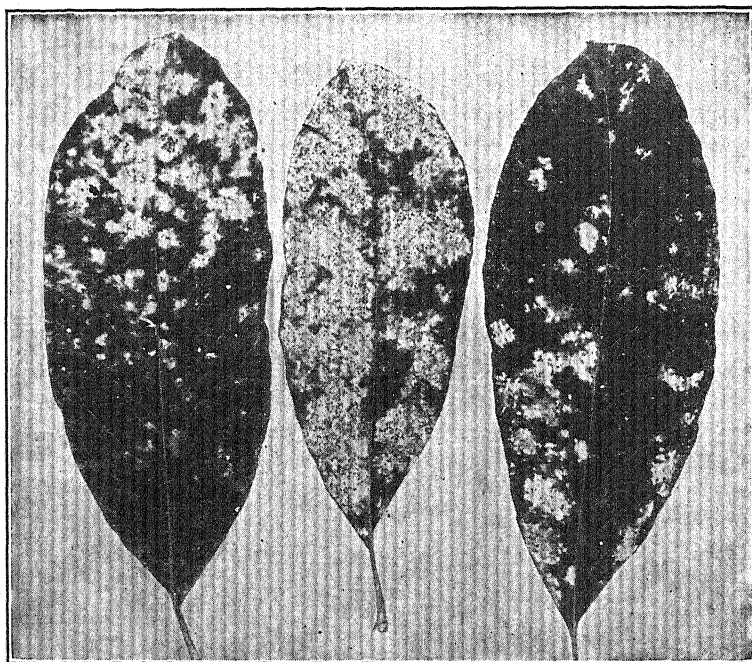


FIG. 79. — Powdery mildew (*Uncinula salicis*) on willow leaves.

reproductive stages, on the surface of the host plant, although in some cases marked structural changes are wrought in the parasitized tissues. The superficial nature of the mycelium is a striking characteristic of this family of fungi. In all of the powdery mildews, with one exception, the mycelium of the fungus grows entirely on the surface of the host, sending only short haustoria into the epidermal cells of the infested plant. This occurrence of superficial mycelium is one of the characteristics which distinguish the powdery mildews from the downy mildews, the mycelium in the latter being entirely imbedded in the host tissue.



During the early development of the powdery mildew diseases the invaded surfaces take on a whitish or grayish appearance due to the mat of mycelium covering the surface (Fig. 79). This whitish surface also acquires a powdery appearance, almost as if dusted with a coarse white



FIG. 80. — Powdery mildew (*Sphaerotheca mors-uae*) on gooseberries.

powder. This condition is due to the production of immense numbers of the asexual or imperfect fruiting structures. In certain cases the mycelium, after a time, changes to a dark brown color so that the char-



acteristic white appearance is lost. In most cases, however, the mycelial growth remains light in color to the end of the season. The powdery or mealy appearance disappears to a great extent as the end of the growing season approaches and the mildewed surface becomes thickly dotted with small black specks, which are the perfect-stage reproductive bodies of the fungus. These fruiting bodies are barely visible to the naked eye but are more conspicuous when viewed with a hand lens (Fig. 79).

In addition to these superficial signs of the disease consisting of the various structures of the fungus itself, there are frequently exhibited striking symptoms in the form of a decided stunting or deforming of affected parts. Sometimes actual necrosis may result. Symptoms of this sort are found in the case of the apple powdery mildew where the growing shoots may be greatly stunted, the leaves dwarfed, and even the death of the whole shoot sometimes results. In the case of the grape powdery mildew the fruits may be deformed, cracked, russeted and stunted in size. The powdery mildew of roses many times blights the blossoms to such an extent as to render them worthless for ornamental purposes. Badly mildewed leaves of any plant are apt to turn yellow and drop.

**Morphology of the fungus.** — There are half a dozen important genera and numerous species of the powdery mildew fungi but in general the morphology and life history of all of them are much the same with only minor differences. A general discussion of these features will be given here and special cases, especially in the details of the life history, will be taken up under the particular mildew diseases discussed in succeeding pages.

The vegetative mycelium of these fungi is superficial in all cases except the mildew of the hazel and certain other trees, caused by *Phyllostictia corylea* (Fig. 82 D). In the latter case hyphae really penetrate the leaf tissue beneath the epidermis. In all other known cases the mycelium grows over, and is closely appressed to, the epidermal surface, but only haustoria penetrate the epidermal cells and these never extend beneath the epidermis. The fact that the mycelium is superficial in this way has a bearing on the control of powdery mildew diseases.

**Conidial stage.** — In the powdery mildews the asexual or imperfect reproductive stage consists of conidiophores and conidia. The conidiophores are short branches which arise from the hyphae on the surface of the host. These branches stand erect, that is, more or less perpendicular to the surface on which the fungus is growing. The conidiophores themselves never branch. As the conidiophore grows up cross walls cut off cells at the apical end. The end cell breaks off as it matures and becomes a conidiospore, oblong in shape with somewhat rounded

ends, and varying in size with the species, some being as large as  $25-30 \times 15-17 \mu$ , while others are much smaller, even down to  $4-5 \times 2.5-3 \mu$ . In some cases a number of cross walls form in the conidiophore and a chain

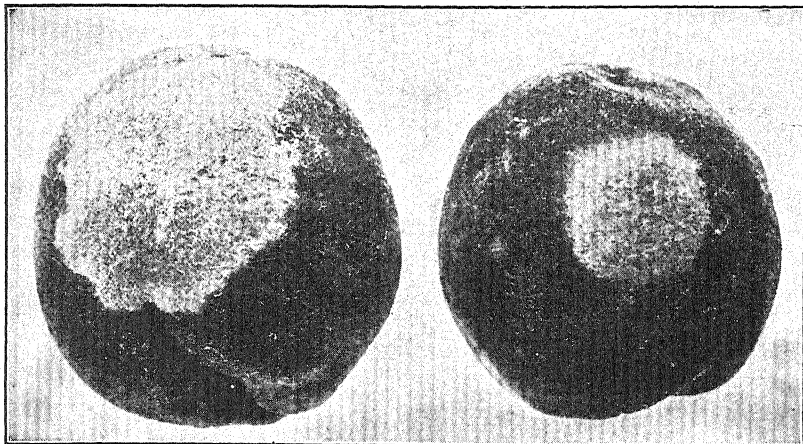


FIG. 81. — Powdery mildew (*Sphaerotheca pannosa*) on peach fruits. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest and Hort. Rept. 1911-12.)

of spores several cells long is produced before any of them separate (Fig. 82 E).

*Perfect stage.* — This stage originates in a sexual process. As the fungus approaches maturity sex organs, oögonia, and antheridia appear on the mycelium. Sexual fusions occur and as a result perithecia containing asci are produced. These are the minute black bodies observed on the mildewed surfaces of plants. They are provided with appendages of various sorts. The perithecia vary in size in the different species and genera and even within the same species. The smaller ones measure about  $50$  or  $60 \mu$  in diameter while some of the larger ones run well over  $200 \mu$  in diameter. The number of asci in a perithecium as well as the number of spores in an ascus is variable. The number of asci and the character of the appendages vary with the different genera of the powdery mildew family. There are six common genera of the Erysiphaceae and in two of these genera there is a single ascus in each perithecium while in the other four genera each perithecium contains more than one ascus. There are four different types of appendages found in the six genera. All of these features are illustrated in Fig. 82, and combinations of these characters are used in the following key which is useful in determining the genus to which any particular powdery mildew fungus belongs.

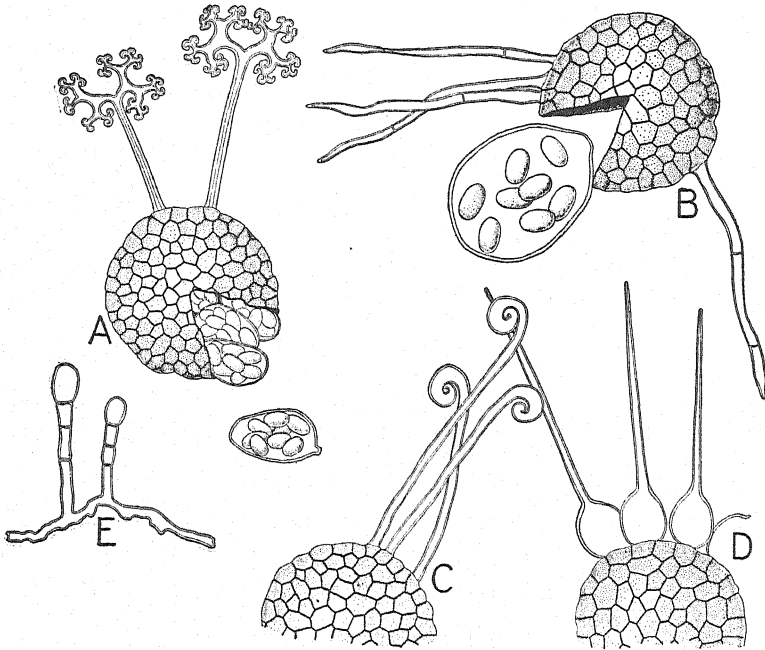


FIG. 82. — Fruiting stages of various genera of powdery mildew fungi. A, perithecium of *Microsphaera* from lilac leaf; B, perithecium of *Sphaerotheca* from gooseberry; C, perithecium of *Uncinula* from willow; D, perithecium of *Phyllactinia* from dogwood; E, conidial stage of a powdery mildew fungus growing on red clover.

- A. Perithecial appendages hooked or coiled at the apex. 1. *Uncinula*
- B. Perithecial appendages rigid, spike-like, with a bulbous base. 2. *Phyllactinia*
- C. Perithecial appendages dichotomously branched at the tip.
  1. Perithecium containing a single ascus. . . . . 3. *Podosphaera*
  2. Perithecium containing several asci. . . . . 4. *Microsphaera*
- D. Perithecial appendages similar to the mycelium, simple or irregularly branched.
  1. Perithecium containing a single ascus. . . . . 5. *Sphaerotheca*
  2. Perithecium containing several asci. . . . . 6. *Erysiphe*

**Life cycle.** — The conidia serve as a rapid means of dissemination during the summer or growing season. They are disseminated largely if not entirely by the wind. Wintering over is effected in most cases by the ascospores in the perithecia. These bodies afford a maximum of protection for the ascospores which are discharged only upon the breaking or disintegration of perithecia in the spring. Apparently in some

cases the mycelium overwinters in the form of dense felts or tufts on dormant twigs or buds of the host plant (Fig. 84). This seems to be true in the case of the powdery mildews of apples, roses, peaches, gooseberries and possibly others. In such cases the mycelium apparently lies dormant during the winter and resumes active vegetative growth again the next spring, spreading over new leaves and shoots and soon producing a crop of conidiospores which then account for widespread dissemination of the fungus during the remainder of the season.

### Powdery Mildew of the Apple

Caused by *Podosphaera leucotricha* (E. & E.) Salm

The powdery mildew of the apple is widely distributed over the world. It is known to occur in various countries of Europe, in Japan, in Australia and New Zealand and is well established in North America throughout the United States and Canada. In the United States it has long been recognized as a nursery disease in the older sections of the country, being considered of little importance in the orchard. In the Pacific Coast states, however, the disease has been recognized as a serious orchard trouble, especially in certain irrigated districts where a large apple-growing industry has sprung up in recent years. No estimate of the losses incurred from this disease is possible but it undoubtedly is of considerable economic importance. In the nursery the damage consists of injury to the leaves and shoots. In the far West similar injury occurs in the orchard and furthermore the disease frequently disfigures the fruits. It is said that the disease seldom occurs on fruit east of the Rocky Mountains. In severe cases the injury to the foliage is so great as seriously to devitalize the trees, thus causing a fruit failure the next year. In addition to the apple this fungus may also attack the pear, quince, plum, cherry, hawthorn and serviceberry, but it is of most importance on the apple. Among apple varieties those most susceptible to the mildew fungus include the Gravenstein, Spitzenberg, Jonathan, Yellow Newtown, Grimes and Rome Beauty. Some of the less susceptible varieties include the Winesap, White Pearmain, Red Astrachan and Rhode Island Greening, but no variety is completely immune.

**Symptoms.** — The white powdery growth, typical of the powdery mildews in general, occurs on apple foliage affected with this disease. A striking characteristic, especially as it occurs in the orchards of the Pacific Coast states, is the large number of mildewed shoots which are greatly stunted in growth, the internodes being shortened and the

leaves dwarfed and rolled (Fig. 83). During the dormant season when the leaves are off, many of these shoots can be found on the tree, still covered with whitish mycelial felts of the causal organism (Fig. 84).



FIG. 83. — Apple shoots affected with powdery mildew (*Podosphaera leucotricha*). (After Jackson, Ore. Agr. Exp. Sta. Crop Pest and Hort. Rept. 1911-12.)

In some cases perithecia can be found on these dormant twigs but they are sometimes absent or rare. In the same region the fruits as well as the foliage are oftentimes affected. The symptoms on the fruit consist

chiefly of a stunting in growth and a russetting which produces various disfiguring patterns on the skin of the fruit. Blossoms are also sometimes mildewed so that fruit does not set.

**Morphology and life history.** — Most of the apple powdery mildew in the United States is caused by *Podosphaera leucotricha* but another species, *P. oxyacanthae* has occasionally been found on the apple in the eastern part of the country. The perithecia of *Podosphaera* each contain a single ascus with eight spores. The appendages of *P. leucotricha* are mostly unbranched at the tip but a few of them are dichotomously branched. All of the appendages of *P. oxyacanthae* are branched in this manner. The perithecia of the apple mildew fungus apparently play little part in perpetuating the organism. In some cases they are rarely found and in other cases, where they are produced in abundance, extensive investigations indicate that they play little if any part in the overwintering of the fungus and in starting primary infections in the spring. All available knowledge indicates that, in most cases at least, the fungus hibernates as dormant mycelium in the buds and when the buds open in the spring the fungus resumes activity and invades the new leaves and shoots as they emerge from the buds. If the infested bud happens to be a flower bud, the blossoms emerging from it are likely to be invaded by the mildew fungus. Dissemination during the growing season occurs by means of the conidiospores which are scattered by the wind. This fungus is able to thrive in arid climates because of its low moisture requirement

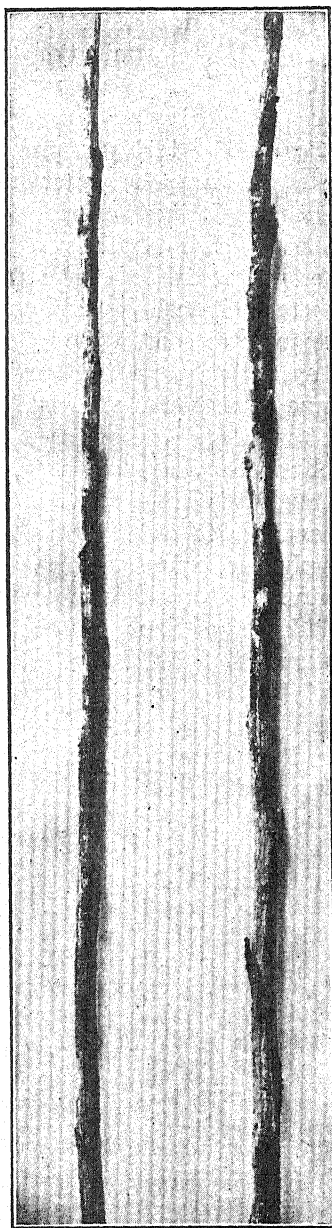


FIG. 84. — Apple twigs in winter, showing dormant condition of powdery mildew fungus. (Photograph by Corsaut, Ore. Agr. Exp. Sta.)

for spore germination. Apparently frequent dews at night are sufficient for spore germination and infection.

**Control.** — There are two chief items in the measures recommended for control of apple powdery mildew: (a) pruning out the mildewed twigs, and (b) the use of protective sprays. Many affected twigs can be detected during the winter pruning operations and should be removed at that time. Since it is manifestly impossible to get all of the fungus in this way, an adequate spray program is necessary. Sulfur in a satisfactory form is the best known remedy for powdery mildew. In regions where apple-scab prevails the regular lime-sulfur scab sprays are usually adequate to control the mildew also. In some arid regions, however, a special program for powdery mildew is necessary. Lime-sulfur or some of the commercial sulfur pastes are to be preferred. During very hot weather, however, so much injury may result from the use of the sulfurs that a copper spray may have to be substituted for sulfur even though it is less effective in controlling the mildew. Fisher (3) recommends the following schedule: 1. First mildew spray, lime-sulfur 1-50, apply just before the blossoms open. 2. Second mildew spray, lime-sulfur 1-50, apply as soon as most of the petals have fallen. 3. Third mildew spray, lime-sulfur 1-50, apply about two weeks after the second spray. 4. Fourth spray, lime-sulfur 1-50, or neutral bordeaux, or sal soda bordeaux, or ammoniacal copper carbonate, apply about two weeks after the third spray. It will be noted that the copper sprays may be used in the last application if the weather is too hot to use the sulfur spray safely.

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### Powdery Mildew of the Grape

Caused by *Uncinula necator* (Schw.) Burr.

This disease has been known in Europe since 1845 and in the United States since 1859. It is now known on every continent with the possible exception of South America. In the United States it is widely distributed throughout the country from the Atlantic seaboard to the Pacific. In certain sections of this country, notably in the Pacific Coast states and in parts of western New York, it is far more serious than other grape diseases such as downy mildew and black-rot. Besides various grape varieties this fungus also attacks the Virginia creeper (*Ampelopsis*).

**Symptoms.** — The disease may affect any part of the plant, leaves, blossoms, fruits and young shoots. On the leaves and green shoots the fungus appears in the usual white patches covering a part or all of the surface. If young canes are severely attacked they are considerably stunted in growth. Mildewed blossoms fail to set fruit. The effect on the fruit varies, depending upon its age when attacked. Fruits attacked when very young may drop. When half-grown or older fruits are attacked the effect is various. The berries may develop irregularly and become hardened. The surface of mildewed fruits is usually roughened and discolored. Cracking of fruit frequently occurs and molds or other saprophytic fungi frequently invade the cracked fruits.

**Life cycle.** — This fungus conforms for the most part to the typical life cycle of the group. Summer spread is effected by means of the conidia. In many cases perithecia are scant or entirely absent. In such cases it is thought that this fungus hibernates in the mycelial condition. At the end of the growing season resistant cells, which are supposed to survive the winter, are formed in the mycelium near the haustoria. In this manner the grape-mildew fungus probably overwinters in the absence of perithecia and ascospores as appears to be the case in several of the important powdery mildews including that on the apple.

**Control.** — The best means of control yet discovered for grape powdery mildew is sulfur dust. This method of control has been used with great success in the vineyards of California. In New York sulfur dust causes injury to the American grapes but can be used on vines of European origin. The number of applications of sulfur necessary to control the disease will vary from one to six, depending on the locality, the season and other conditions. The first application should be made when the shoots are not more than six or eight inches long. A second sulfuring is usually necessary just before or during blossoming. Whether



or not additional applications are necessary depends upon conditions among which are the effectiveness of the first applications, the susceptibility of the variety and the weather conditions. Sometimes a third application is necessary when the fruit is half grown. Additional applications are necessary only in severe cases or as a result of carelessness in the first applications.

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#### LABORATORY STUDY OF POWDERY MILDEWS

**A. Symptoms.** — Examine specimens of various plants affected with mildew. Mildewed specimens from willow, oak, lilac, apple, grape, gooseberry, peach, rose, clover or any other plant available may be used. Note the white powdery appearance in the early stages. What causes the powdery effect? Use binocular or low power of regular microscope to examine surface of specimens. Can you see mycelium and conidiophores? On material collected late in the season find the ascocarps. On dormant apple or peach twigs or rose canes observe the thick felty growths of dormant mycelium. On nearly mature gooseberries note how dark in color the mildew becomes. If grapes affected with powdery mildew are available note the stunting and cracking of the fruits. The dwarfing effect can be seen on the new shoots of apple trees where mildew infection occurs early. Make habit sketches to illustrate any of these symptoms observed.

**B. Morphology and life history.** — The powdery mildew fungi produce both conidiospores and ascospores.

1. *Conidial stage.* — If good material is available both the vegetative mycelium and the conidial stage can be seen to advantage if proper precautions are used in making the mounts. Simply scraping off the mildew from the leaf and mounting in water is not a very satisfactory way of making mounts. Good mounts of mycelium with conidiophores and conidia attached can be made if a drop of dilute KOH is placed on the mildewed surface for a minute and then this treated surface is scraped with a scalpel and the scrapings mounted in water as usual. Draw mycelium and conidiophores.

2. *Perfect stage.* — Scrape some of the ascocarps from a leaf and mount in the usual manner. Note the appendages. Break open the perithecium by gently pressing on the cover glass and count the asci and ascospores which escape. Refer

to the key on page 304 and determine to which genus this species belongs. Draw a perithecium showing appendages and escaping asci. Draw one ascus with its contained ascospores much enlarged. Examine perithecia from willow, oak, apple, rose, gooseberry, grape, or any others available, and, by means of the appendages and the number of asci per perithecium, determine the genus to which each belongs.

3. *Life cycle*. — What part does each spore stage play in the perpetuation and dissemination of the powdery mildews. Look up the life history of apple mildew, grape mildew and others. In any case do you find special methods of overwintering other than by one of the spore forms?

C. *Notes*. — In writing the notes for this exercise, include not only a general discussion of the morphology and life history of the powdery mildews in general, but give a special discussion of the powdery mildews of apples and of grapes.

#### REVIEW QUESTIONS

1. Describe the symptoms and signs of powdery mildew as it appears early in the season and also its appearance as the growing season draws to a close. Describe the effects on the fruits of apple, grape, and gooseberry. What are its effects on roses, especially the blossoms? Describe its appearance on dormant twigs of apple, peach or rose bushes.

2. What is the most striking characteristic of the vegetative mycelium?

3. How many fruiting stages are possessed by the powdery-mildew fungi? Describe each.

4. What spore stage is responsible for the summer spread? The overwintering of the fungus? What other method of hibernation is exhibited by some of the powdery mildews?

5. What fungicide usually gives the best results in control of these diseases?

6. What is the host range of powdery mildews in general?

7. What is the nature of the damage done by these fungi?

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## Scab (*Fusarium*-blight) of Cereals

Caused by *Gibberella saubinetii* (Mont.) Sacc. and *Fusarium* spp.

Under this heading will be discussed a group of troubles which affect the small grains, wheat, oats, barley and rye, and also certain manifestations of disease in corn due, in part, to the same organisms. On the small grains the earliest recognized phase of this trouble has been known for many years as "scab" or "wheat-scab." This consists of a typical blighting of the heads of wheat and the other small grains, which has more recently been termed "*Fusarium*-blight" (3), because the malady is now known to include a seedling blight and other manifestations of disease in addition to the blighting of the heads to which the name "scab" was originally applied. In corn a seedling-blight also occurs as well as the serious root-, stalk-, and ear-rot troubles due, in part at least, to the same organisms concerned in the *Fusarium*-blight of wheat and the other small cereals. There are other corn-rot troubles and also root-rot diseases in the small cereals which are caused by other organisms, such as the *Diplodia* rot (6) of corn and the *Helminthosporium* root-rot of wheat (7) which will not be included in this discussion.

The wheat-scab trouble has been reported from Europe, Asia and Australia as well as from the North American continent. In the United States it occurs in nearly every state, particularly east of the Rocky Mountains, and is especially prevalent in the big cereal growing sections of the central and eastern United States. The corn root-, stalk- and ear-rot troubles are serious in the corn belt including such states as Iowa, Illinois, Indiana, Ohio and Kansas. They also occur in the South. Losses from these diseases are difficult to estimate. In 1921 The Plant Disease Survey (19) estimated a reduction of over 10,000,000 bu. in the wheat crop of the United States due to scab. During the same year the loss in the corn crop due to root- and ear-rots was placed

at approximately 169,000,000 bu. During 1922 the same authority (20) estimated the losses due to corn root- and ear-rots at over 120,000,000

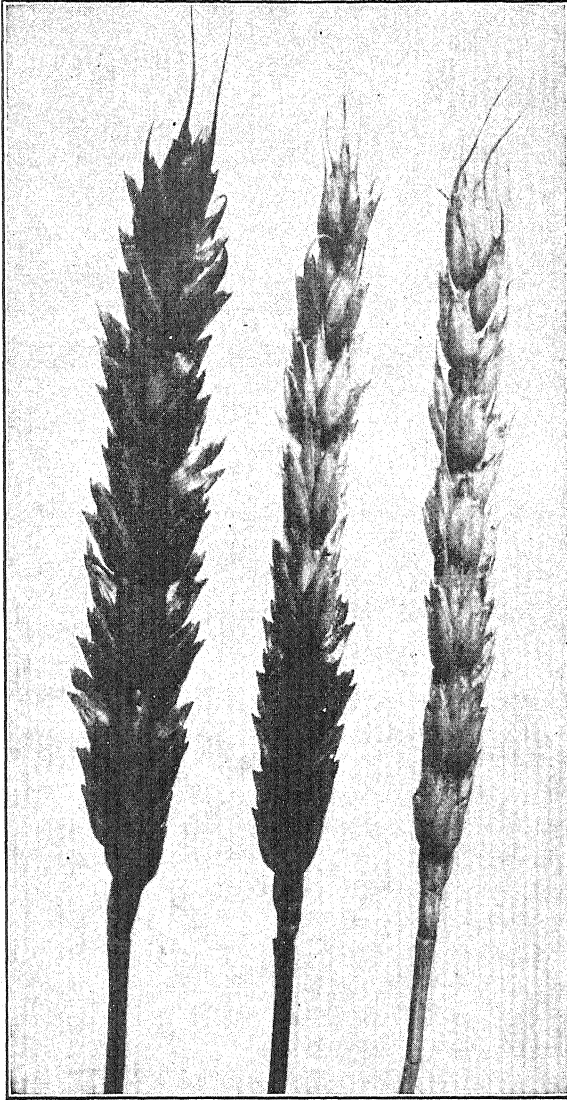


FIG. 85. — Marquis wheat heads showing blight produced by *Gibberella saubinetii*. (Photograph by J. G. Dickson.)

bu., while wheat-scab took a toll of 6,000,000 bu. In 1923 (21) wheat-scab caused a loss of 8,642,000 bu., while corn root- and ear-rots caused

a reduction of 205,751,000 bu. These estimates for the corn-rot diseases presumably cover all types of root- and ear-rots but undoubtedly a large percentage of the losses was due to those rots related to the wheat-scab trouble.

**Symptoms on wheat.** — The characteristic symptoms of wheat-scab as it was originally known were confined to the heads of the host plant. The disease is now known not only to blight the heads but it also affects the seeds and results in a seedling-blight and a root-rot or foot-rot, and sometimes even attacks the stems. On the heads of wheat the blight may attack at any point, usually affecting only one spikelet at first. It may later spread to other spikelets if conditions are favorable, or remain confined to a single spikelet. In severe cases the whole head may be involved. Occasionally it extends down the stem to the first node. The first indication of infection consists of watersoaked areas, slightly brown in color, on the glumes. As the disease progresses the affected areas dry out and take on a ripened appearance (Fig. 85). If the infection spreads into the rachis at the base of the spikelet and completely girdles it the portion of the head above this infected region will die and dry up even if it is not directly invaded by the fungus. After a while a cottony fungous growth, slightly pinkish in color, appears on the dead surface. This growth becomes evident first at the point of infection but later may spread farther over the infected area. Conidia develop on this growth (Fig. 86) and with age the pink tint turns to a darker salmon color. The pinkish conidial masses are more apt to form at the bases of the spikelets where moisture is held for a longer time. On the other small grains, rye, barley and oats, the symptoms are quite similar to those on wheat with minor variations.

In the blighted heads the grains themselves are frequently invaded, resulting in lightweight, shriveled kernels. If diseased seeds are planted the result is disastrous. In some cases the seedlings never emerge. The embryo may be invaded and killed before the plumule and roots elongate. Infected parts of the kernel become reddish brown to carmine red (5). In other cases the seedling may emerge and struggle along for some time, succumbing sooner or later, depending upon the severity of the attack. In some cases the roots may be badly rotted and the seedling dwarfed, but such plants, under favorable weather conditions, may send out new roots and partially or wholly recover. Other fungi besides those included in the heading of this section cause root-rots of cereals (7).

**Symptoms on corn.** — Several different types of symptoms are produced on corn by *Gibberella* and species of *Fusarium*, including a seedling-blight, a root-rot, a stalk-rot and an ear-rot. Seedling-blight

results when corn grains which are more or less infected with the rot organisms are planted. The fungi may grow directly from the seed grain into the seedling or spread into the roots. Badly infested seedlings

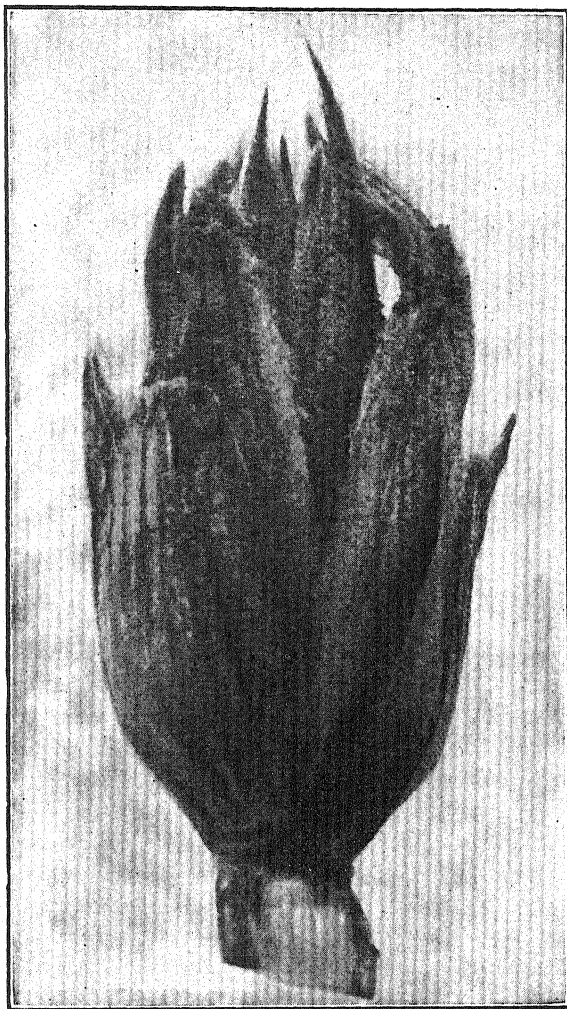
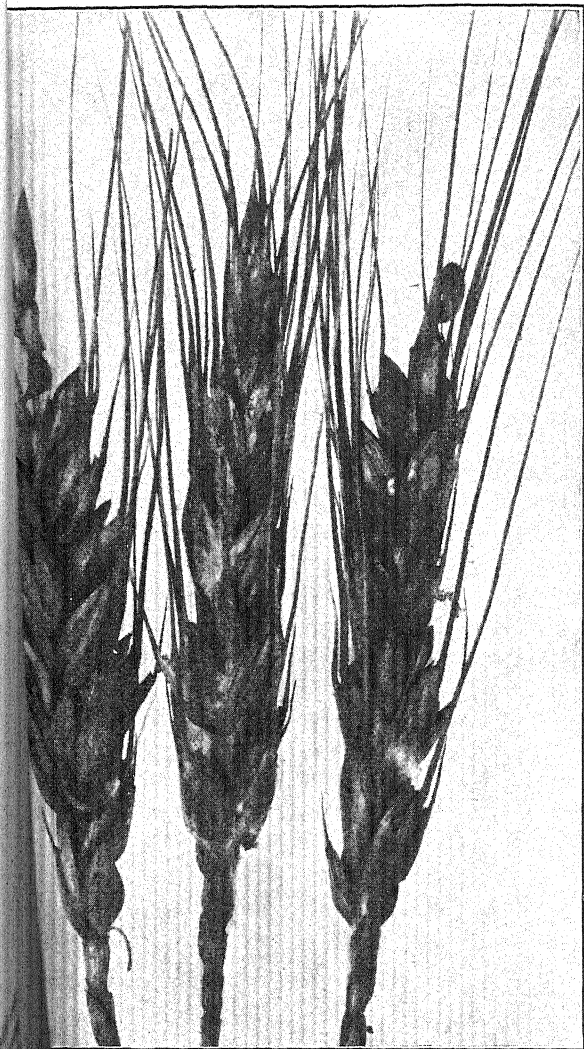


FIG. 86. — Conidial stage of *Gibberella saubinetii* on wheat spikelet. Greatly enlarged. (Photograph by J. G. Dickson.)

die soon after emergence or in severe cases they may never appear above the ground. Slightly infected seedlings may partly recover but seldom develop into normal plants. Plants with partially rotted root systems naturally do not make a normal growth and show the stunting and

## DISEASES CAUSED BY FUNGI — ASCOMYCETES

throughout their life. They cannot withstand unfavorable conditions as well as healthy plants. The fungus



Turkey wheat showing perithecial development of *Gibberella* (by J. G. Dickson.)

Stalks and weaken the plants further. Root-rots and cause lodging. Some stalks break while others may be breaking. Badly diseased plants may be barren. Less



severely affected plants may bear ears of small size or inferior quality. Sometimes the ear shanks are invaded and break down. Often the fungus invades the ear and causes an ear-rot.

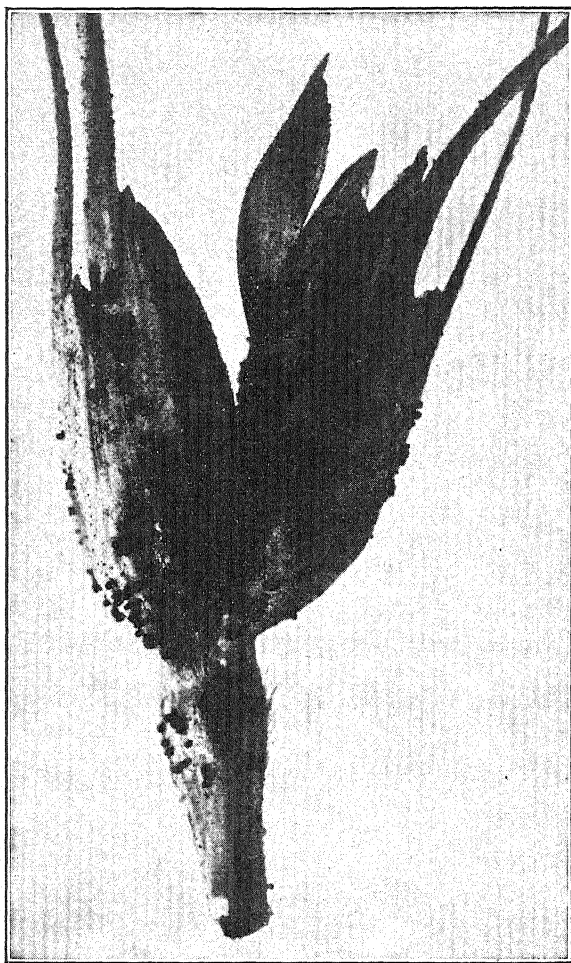


FIG. 88. — A single spikelet of wheat showing perithecia of *Gibberella saubinetii*. Much enlarged. (Photograph by J. G. Dickson.)

**The causal fungi.** — Most of the scab or Fusarium-blight of the small grains and some of the corn rots described above are caused by *Gibberella saubinetii*. The conidial or imperfect stage of this fungus belongs to the form genus, *Fusarium* (see Chapter XX), and as such is known as *Fusarium graminearum*. In addition to this species, several other



imperfect fungi, species of *Fusarium*, are sometimes found associated with this group of troubles on both wheat and corn. Some species found in blighted plants of the small grains include *F. culmorum*, *F. herbarum*, *F. avenaceum* and *F. solani*. On corn, *F. moniliforme* is one of the most frequently encountered species. Thus it is seen that several species of *Fusarium* are concerned as causal agents in this group of diseases but that *Gibberella saubinetii* is the chief one connected with these wheat troubles and is also frequently important on corn, apparently attacking both these crops with equal facility. (References to other fungi which cause root-rots or ear-rots in wheat and corn will be found at the close of this section.)

The fungus, *Gibberella saubinetii*, produces two spore forms in its complete life cycle, namely, conidia and ascospores. The first type of spore to appear consists of the conidia which are borne in abundance

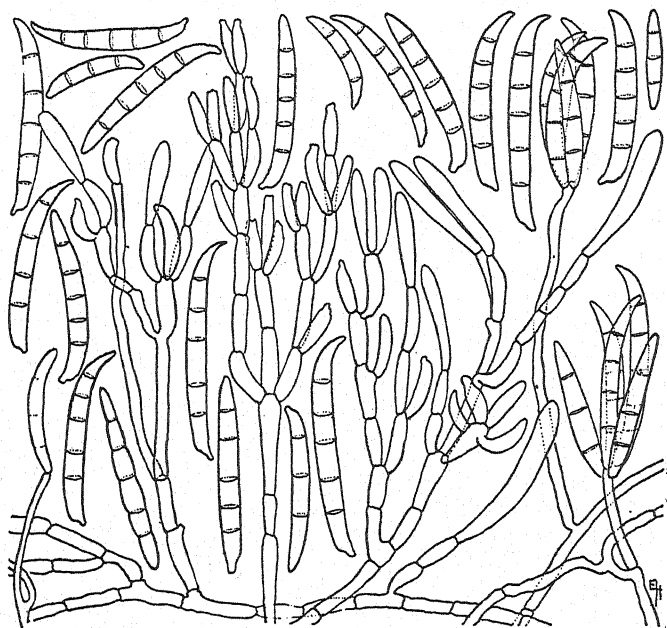


FIG. 89. — Conidiophores and conidia of *Gibberella saubinetii*.

on the surface of diseased parts where the pinkish coating of fungous growth occurs. These conidiospores are long, slender, curved, septate spores, typical of the form genus, *Fusarium*, in which this fungus was classified before its perfect stage was discovered. The conidiospores range in size from  $35 \times 4 \mu$  up to  $75 \times 5.5 \mu$  with the great majority

coming within a size range of  $45-65 \times 4.2-5.5 \mu$ . Most of them are 5-septate with occasional spores having as few as 3 or as many as 6 or 7 septa (Fig. 89).

The perfect stage occurs on dead, blighted heads of wheat (Fig. 87), or the other small grains, and on dead corn-stalks. The perithecia are small black bodies (Fig. 88), occurring singly or in groups, which rest on

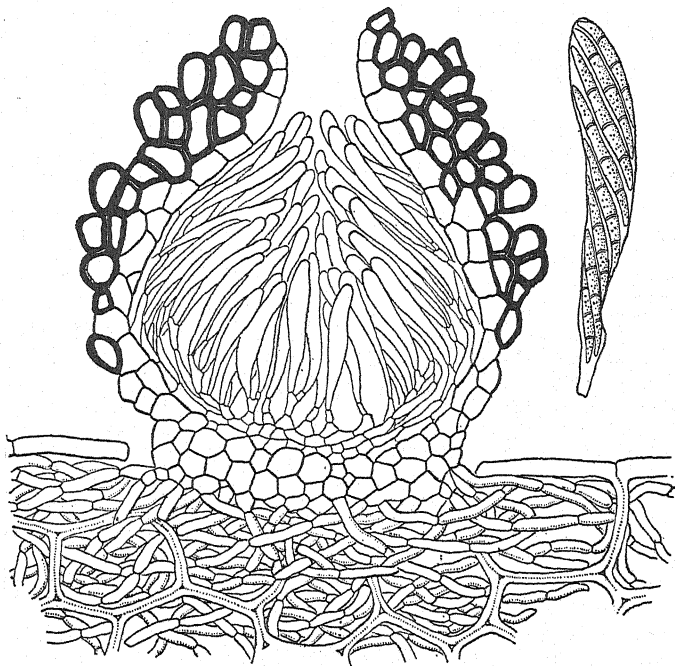


FIG. 90. — Section of a perithecium of *Gibberella saubinetii*. At the right a single ascus enlarged.

the surface of the host or may be more or less imbedded in the mycelial crust where conidia have been produced. They are ovoid to subconical in shape and measure  $150-250 \times 100-250 \mu$  (Fig. 90). The asci may number over a hundred to the perithecium and contain 8 ascospores each. The latter are fusiform, slightly curved, mostly 3-septate and measure  $20-30 \times 3.75-4.25 \mu$  (Fig. 90).

**Life cycle.** — The conidiospores of *Gibberella saubinetii* as well as of the other species of *Fusarium* associated with these troubles are produced abundantly on the surface of diseased parts. They may be found on the underground decayed parts of blighted seedlings, on blighted heads, on diseased stems and on old straw and cornstalks left

in the fields. It has been shown (3) that the mycelium remains alive in these old cornstalks and straw all winter and produces new conidia in the spring. Other plant débris also may serve as an overwintering medium for this fungus. The perithecia of the perfect stage are likewise produced on any infected part following conidia-production, and this ascosporic stage plays an important part in the overwintering of the fungus. The organism is also carried over in diseased seed. Dissemination of conidiospores is effected to some extent by the wind. Rain plays an important rôle in softening the clumps of conidia and splashing them about. Ascospores probably depend largely on the wind for dispersal. Infection on the heads of wheat and the other cereals occurs at blossoming time or later. Seedling-blight of course results from the use of diseased seed. This holds true for corn as well as wheat. Root, stalk and ear infections on corn may take place locally throughout the growing season or the disease may persist in slightly blighted seedlings which partially recover and struggle along in a weakened condition through the season.

There are some practical aspects of the relation of wheat-scab to the corn root-rot which should receive especial mention. Observation and experimentation (9, 15) have revealed a very close relationship between these two troubles. When corn and wheat are rotated successively on the same land there is noted a striking increase in the amount of scab and root-rot. The fungus, *Gibberella saubinetii*, thrives apparently equally well on both hosts and when wheat follows corn or corn follows wheat in close rotation the fungus multiplies rapidly and a great increase in the amount of disease on both hosts occurs.

**Control.** — The fact that the fungi causing these diseases perpetuate themselves on so many different host plants and even on plant débris in the soil makes control very difficult and no perfect preventive is known. The following recommendations, however, if followed, should result in a reduction of losses from this source. (a) Use clean seed. Wheat should be carefully screened to remove all lightweight kernels. Careful selection of seed from disease-free fields, where possible, should be practiced. Seed corn should be selected in the field with the greatest care to insure seed from perfectly healthy plants. All seed corn should be given the germination test before planting to still further insure freedom from these rot organisms (8, 11, 12). (b) Crop rotations should be planned so as to carry a non-susceptible crop between susceptible cereal crops. This is especially true of wheat and corn which should never immediately follow each other in the succession. In case such a rotation is not possible the wheat crop should preferably precede rather than follow the corn crop. (c) All varieties of grain are not

equally susceptible and the development of resistant strains may offer a satisfactory solution of the control problem.

#### LABORATORY STUDY OF WHEAT-SCAB

**A. Symptoms.** — Examine affected heads of wheat and the other cereals, and also seedlings blighted by the scab fungus. On the heads note that individual spikelets may be killed, or that larger portions, even whole heads, are sometimes killed. Find specimens showing the pinkish or salmon-colored growth of the fungus on affected parts. Can you find the fungus on parts of the plant other than the head? How about the grains, and also the stems and roots? Also examine corn plants showing root, stalk or ear rot due to *Gibberella*. Corn seedlings, also, may be blighted by this fungus. On old dead wheat heads and corn stalks find the dark-colored perithecia of the fungus. Make drawings to illustrate any symptoms observed.

**B. The fungus.** — This fungus, *Gibberella saubinetii*, has both an imperfect and a perfect stage represented in its life cycle.

1. *Conidial stage.* — The conidiospores are found in the pinkish fungous growth on the surface of affected parts. Make mounts of these spores. Note their characteristic shape and size. These imperfect stage spores are characteristic of the form genus *Fusarium*. (See text on wheat-scab for mention of other species of *Fusarium* which cause plant diseases; also miscellaneous references at end of Chapter XX.) Draw conidiospores.

2. *Perfect stage.* — The ascospores are found in the perithecia occurring on dead plants. Examine them with hand lens or binocular. Are they deep-seated or resting more or less superficially on the surface of the host? Study sections under compound microscope. Note the characteristic color of the cells in the wall of the perithecium. Draw section to show asci and ascospores in place. Draw a single ascus and contained spores, much enlarged.

3. *Perpetuation, dissemination and infection.* — Which stage of the fungus would you suspect to be the overwintering stage? Look up this question and find out if it overwinters in any other form. How is the fungus disseminated? Where, when, and under what conditions does infection occur? Learn the complete life history of the fungus.

**C. Notes.** — Write complete notes on this disease, giving particular attention to the various symptoms on both wheat and corn and to the life history of the fungus. Discuss control in detail, being careful not to overlook the relation of wheat and corn crops in the rotation system

#### REVIEW QUESTIONS

1. Name the cereals which are subject to attack by *Gibberella saubinetii*.
2. To what form genus does the conidial stage of this fungus belong? Name other species of fungi which cause similar trouble on the cereals.
3. Describe the symptoms of *Fusarium*-blight or "scab" on wheat. What are the symptoms on corn?
4. At what temperatures does the disease develop best on wheat and on corn? (See Chapter V of this text and reference 5 at the end of that chapter.)
5. What does the wheat-corn rotation have to do with control measures for this disease? Why is this so?

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**Black-rot, Canker, and Leaf-spot of Pomaceous Fruits**

Caused by *Physalospora malorum* (Pk.) Shear

This is one of the widespread and well-known diseases of apples and other pomaceous fruits in the United States. While on the whole it cannot be considered by any means the most serious disease of the apple, there are sections of the country where it assumes considerable economic importance. It is a disease which attacks various parts of the host, causing a canker, a fruit-rot and a leaf-spot. Various names have been applied to the different phases of the disease. The fruit-rot has been termed brown-rot, ring-rot, blossom-end rot and black-rot. The last name is now in common use to designate the disease as it occurs on the fruit. The leaf lesions have been called leaf-spot, brown-spot and frog-eye. The last is descriptive of the leaf symptoms and seems worthy of general adoption. The lesions on the bark have been named apple canker, black-rot canker, and New York apple tree canker. The last has been used extensively and is favored by some plant pathologists. However, there does not seem to be any good reason why the term "black-rot canker" should not be used since it fits in with the generally accepted name for the fruit-rot.

**Historical.** — The first record of this disease in the United States was made in 1879 by Peck (24) who reported its occurrence as a serious fruit-rot of the apple in New York state. Within a few years following the first report of the trouble in this country it had been observed in several other states. In 1885 Arthur (2) reported a black-rot on quince fruits in New York. Halsted (13) recorded the occurrence of black-rot on quince fruit in New Jersey in 1892. The disease was reported on apples in Kentucky in 1895. In 1902 Clinton (8) stated that black-rot of apples had been known in Illinois since 1879. The leaf-spot phase of the disease was recorded as early as 1892 by Alwood (1) who reported it from Virginia. In West Virginia the leaf-spot disease was well established in certain sections as early as 1900 according to Corbett (9). The leaf-spot on apple leaves had been observed in the Ozarks before 1908 (27). The canker stage of the disease was reported by Waite in 1898 and by Paddock in 1899, the latter stating that he had observed the disease in New York in 1891. Black-rot cankers were reported from Delaware in 1901, Connecticut in 1906, and Canada in 1909. Within recent years more extensive studies of the disease have been made, especially as to the life history of the causal organism, the host range and control measures. In New Hampshire, I. M. Lewis (18) and Brooks and De Meritt (6) conducted extensive investigations on the leaf-spot phase of the trouble. In the Ozarks, Scott and Quaintance,

and Scott and Rorer (27), worked especially on the control of the disease. In 1915 Craybill (10) published the results of work on the leaf-spot. In 1916 Hesler (17) published the most complete treatise yet put out on this malady, covering all phases of the disease with a complete discussion of the life history of the causal organism.

Outside the United States the disease has been reported from Canada, from various countries of Europe, and from South Africa. The disease has been known in Italy since 1890 and in France (19) since 1901. Berkeley (3) reported an apple-rot in England as early as 1836, which is probably identical with the black-rot. Arnaud discussed the canker disease as it occurs in France and reported the perfect stage of the fungus in 1912. The disease probably was extensively disseminated in Canada previous to 1909. Evans (11) reported the presence of the malady in South Africa in 1910.

**Distribution.** — In the United States the black-rot disease in its various phases is widely distributed, especially in the region east of the Rocky Mountains. According to various sources of information including the bulletins issued by the Plant Disease Survey of the United States Department of Agriculture it is present to a greater or less degree in nearly all apple-growing sections of North America but is of little or no economic importance in the far West although it has been reported as present in the Pacific Coast states. Neither is it of equal importance in all sections in the eastern half of the continent where it is more prevalent. In some regions the canker phase is of more importance than the fruit-rot or the leaf-spot. In other sections of the country the fruit-rot seems to be of more widespread occurrence. While the leaf-spot is quite generally prevalent east of the Rocky Mountains it is only in certain localities that it assumes a dangerous aspect. In certain states, as Oregon, for example, the disease in all of its phases is of very little economic importance. In Canada the disease is of general occurrence in Ontario, Quebec and Nova Scotia. In Europe it is known to occur in England, Russia, Germany, Austria, Switzerland, Holland, Belgium, France and Italy. It also occurs in South Africa and Australia.

**Hosts.** — This disease is of economic importance only on the pomaceous fruits although the causal fungus has been observed on a large number of forest trees and shrubs. It reaches its most serious proportions on the apple but it also affects the pear, the quince and the crab apple. On forest trees and other plants the fungus apparently is a saprophyte living upon twigs and dead bark which have been killed by some other agency. Among the trees and shrubs which harbor the fungus in this way may be listed the alder, ash, elder, dogwood, linden,

hawthorn, lilac, maple, mulberry, oak, pine, rose, witch-hazel and sumac.

Among apples the summer varieties are said to rot more seriously at the time of ripening while greater losses in the winter varieties occur in storage. With respect to the black-rot canker, some of the more susceptible varieties listed in various parts of the country include Twenty Ounce, Spitzenberg, Baldwin, Wagener, Rhode Island Greening, Tompkins King, Ben Davis and Northern Spy. Some of the varieties which are most susceptible to leaf-spot include Twenty Ounce, Rhode Island Greening, Chenango, Baldwin, Black Twig and Ben Davis.

**Economic importance.** — As already indicated the injury caused by the black-rot fungus is of three types, the fruit-rot, the leaf-spot and the canker. Not all of these are of equal importance in all sections of the country where the disease occurs. The fruit-rot is serious in parts of New England and the Middle Atlantic states, and in some mid-west sections including Ohio, Indiana, Illinois and the Ozark region. Serious losses from fruit-rot are also reported from some of the more southern states as Kentucky, Tennessee and the Virginias. Less serious losses from black-rot result in many other states east of the Rocky Mountains. On the whole the leaf-spot phase of this disease is not serious except in a few states. In order to cause serious damage the attacks on the leaves must be severe enough to cause considerable defoliation. In Pennsylvania, Virginia and West Virginia the leaf-spot disease is reported as doing great damage, sometimes causing complete defoliation. It occurs in less severe form throughout the range of the black-rot disease. The canker caused by the black-rot fungus has been reported as serious in Eastern Canada and in New York. In most of the earlier publications the black-rot-canker fungus is credited with being a virulent parasite. However, a few (26) have contended that the fungus apparently follows winter-injury and other injuries to the bark, such as that caused by fire-blight, and is not, in most cases at least, the original cause of the injury. This opinion is now quite generally accepted so that as far as the canker phase of the disease is concerned it is regarded as occurring largely only where some other previous injury has opened the way for the entrance of the black-rot fungus.

The figures given below on the actual losses due to this disease are taken from the Plant Disease Reporter issued by the Plant Disease Survey (Supplements 24, 30, 36, 43) and apparently include losses due to all phases of the disease combined. The Reporter estimates the reduction in yield due to this disease as follows: In 1921 the total loss in the United States was 624,000 bu. In 1922 the loss in New York state alone was 1,488,000 bu.; in Ohio 677,000 bu.; and in the entire



United States 3,941,000 bu. In 1923 the total loss in this country was 2,210,000 bu., while in 1924 the total was 2,651,000 bu.

**Symptoms.** — The first symptom to appear on the fruit is a small brown rotten spot. As the spot enlarges the skin of the apple sooner or later turns black. If the fruit is ripe or nearly so before it is attacked the entire fruit may be involved before the surface becomes black but in

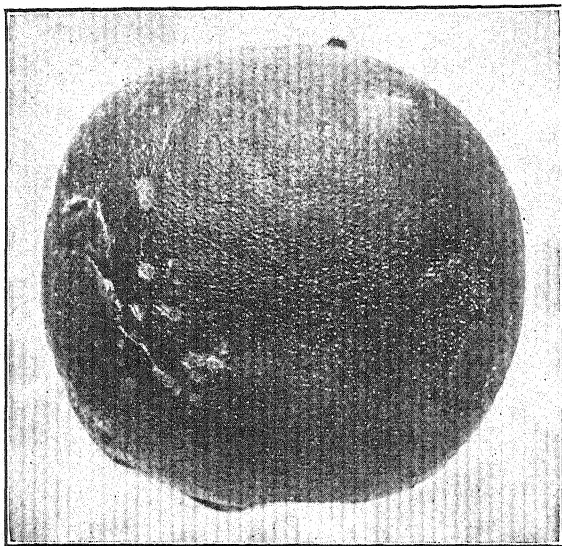


FIG. 91. — Apple fruit affected with black-rot, showing the blackened skin and the numerous pycnidia on the surface. (Photograph by C. R. Stillinger, Ore. Agr. Exp. Sta.)

case green fruits are affected the surface of the rotten area may turn black before the spot becomes very large. The rotting fruit remains plump and firm in appearance until all the flesh is involved in the decay then it finally becomes shriveled and wrinkled. Usually before the rotting fruit reaches the shriveled state the surface becomes thickly dotted with the minute black pycnidial fruiting bodies of the fungus (Fig. 91). The fruit finally dries up completely and becomes mummified. The black-rot mummies can readily be distinguished from the true brown-rot mummies, which also turn black, by the presence of the black pycnidia previously mentioned, which are never present on fruits affected by the latter disease.

The leaf symptoms consist of dead spots in the mesophyll of the leaf. The spots first appear early in the spring shortly after the leaves emerge from the buds. They become evident as minute purple spots which

enlarge rapidly, reaching a diameter of several millimeters, averaging about 4 or 5 mm., but rather indefinite in outline. Later the spot becomes yellowish-brown in color and more definitely delimited from the

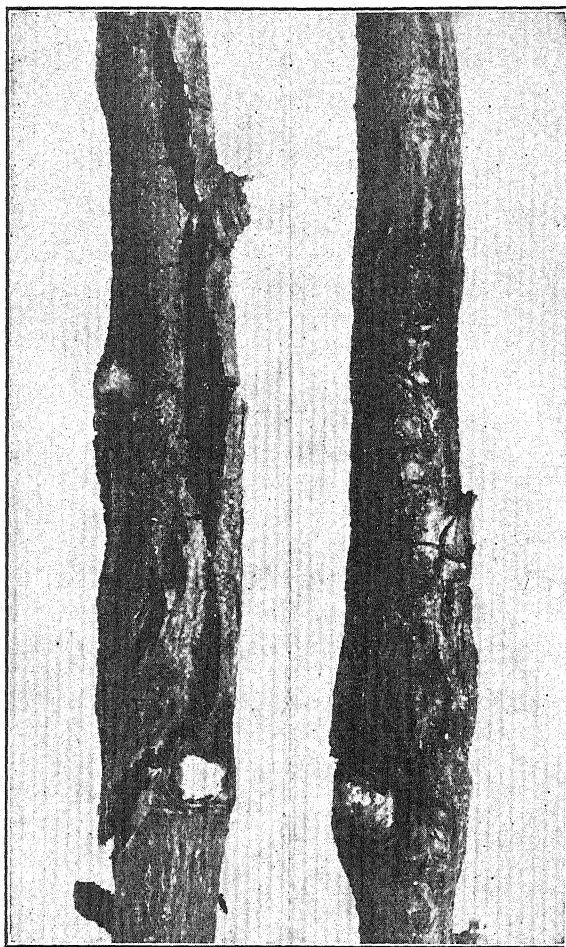


FIG. 92. — Black-rot cankers on apple branches.

remainder of the leaf. After remaining in this condition for a few weeks some of the spots begin to enlarge again in an irregular manner forming lobes or more or less definite concentric rings. The peculiar appearance of the spots at this stage has given rise to the term "frog-eye" (Fig. 93), which is now extensively used to designate this type of leaf spot.

The first symptoms of canker formation consist of slightly sunken

reddish-brown spots. As the diseased area slowly increases in size the color becomes darker. The canker may never attain very great size or it may enlarge from year to year and finally become several feet long. Before the canker is very large a crack or crevice develops at its margin.

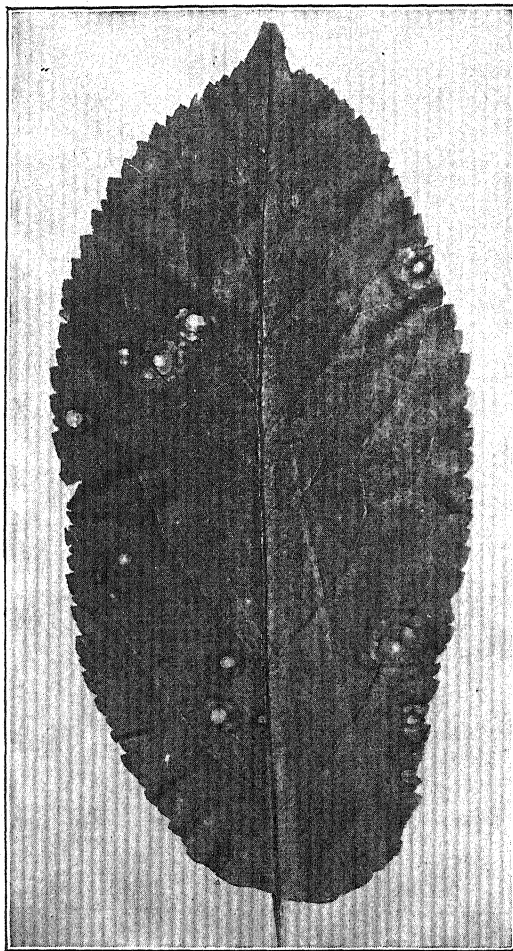


FIG. 93. — Apple leaf showing the frog-eye leaf-spot phase of the black-rot disease.

Later the fungus may extend beyond this margin and kill additional areas of bark, either producing an irregular lobed effect or giving rise to concentric crevices in the bark. The dead bark remains in place for a year or more but eventually sloughs off exposing the dead wood. By the time the canker is six months to a year old, pycnidia of the fungus

similar to those on rotted fruit become evident over the surface of the dead bark (Fig. 92). Small branches may be girdled the first year, or larger limbs after a longer time, if the fungus continues to spread year after year. Sometimes the canker may entirely cease to enlarge before the branch is completely girdled.

**Morphology and life cycle of the fungus.** — The causal organism of black-rot is *Physalospora malorum*. Two fruiting stages are known in the life history of this fungus, an imperfect or pycnidial stage and a perfect or ascogenous stage. The pycnidia are found on the fruit, bark and leaves of the invaded plants. These fruiting bodies vary a great deal in shape and size, and occur singly or occasionally two or more united in a stroma. A typical simple pycnidium found in the bark of an apple twig is illustrated in sectional view in Fig. 94. A fruiting body of this type may measure on the average from 200 to 300  $\mu$  in diameter. The pycnosporos or conidia are borne within this flask-shaped body on short stalks or conidiophores. The pycnosporos vary greatly in size, shape, color and septation. They range in size from 7 to 16.2  $\mu$  broad by 16 to 36  $\mu$  long, the average being about 12 by 25  $\mu$  (17). Their color

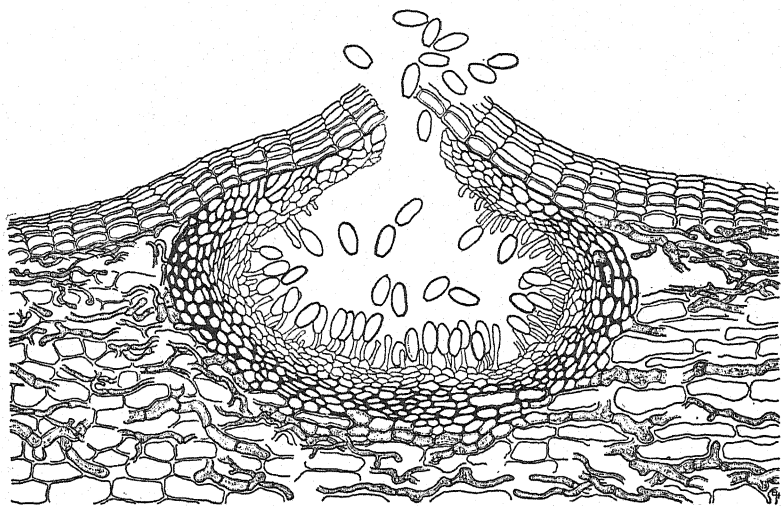


FIG. 94. — *Physalospora malorum*. Section of pycnidium in apple bark, showing conidiophores and conidia.

is at first hyaline, becoming brown with age. In shape the spores are typically ellipsoidal (Fig. 94), but vary from globose to pear-shaped or oblong. The pycnosporos are more frequently one- or two-celled, but occasionally three- or four-celled spores are found. A pycnidium may

contain only one-celled spores or only two-celled spores, or again one may be found containing both.

The perfect stage has been found a few times by Hesler and others on various hosts including apple, witch-hazel and oak. Apparently this stage is of infrequent occurrence and has little economic importance in the life cycle of the fungus. The fruiting body of the ascigerous stage is a perithecium which is shaped somewhat like a typical black-rot pycnidium and imbedded in the outer bark with the ostiole emerging through the outer cork layer at maturity (Fig. 95). The average size of a perithecium is about  $225\ \mu$  in the vertical diameter by  $325\ \mu$  in the horizontal diameter. The asci are clavate to cylindrical and measure on the average about  $26$  by  $155\ \mu$ . The ascospores are approximately ellipsoidal in shape and average  $11.5$  by  $28\ \mu$  in size.

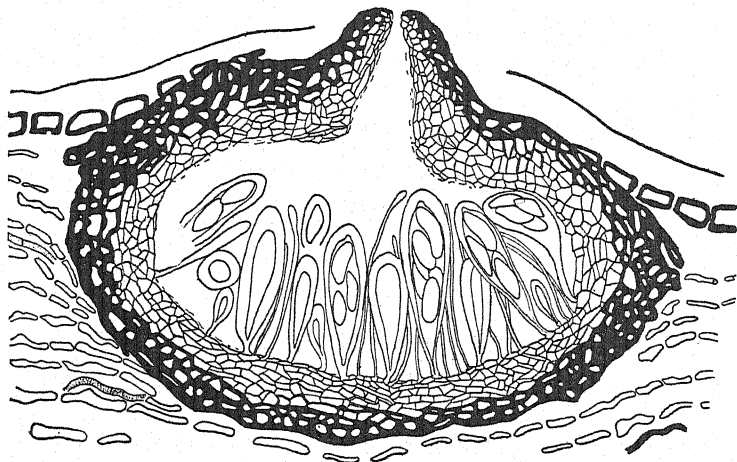


FIG. 95. — *Physalospora malorum*. Section of perithecium showing asci and ascospores. (After Hesler, Cornell Univ. Agr. Exp. Sta. Bul. 379.)

The fungus hibernates as mycelium or as pycnospores in the affected bark, in mummified fruit and also possibly in fallen leaves. The pycnospores are discharged during the spring and summer and disseminated by the wind, rain and possibly insects. Temperature and moisture conditions seem to determine the exact time of spore discharge. A temperature of  $60^{\circ}\text{F}$ . or above and considerable humidity seem necessary conditions for the opening of the pycnidium and the discharge of spores (17). New infections on foliage have been noted from May until September. Infections on fruits occur mainly as the fruit approaches maturity or even in storage. Apparently some sort of wound or injury is necessary before infection can occur on either bark

or fruit. Some writers state that wounds are also necessary for leaf infection while others state that no wounds are necessary in this case.

**Control.** — The control measures for this disease consist chiefly of two practices, namely removal of the sources of inoculum and protection of susceptible parts by fungicides. Since most of the spores which cause new infections come from the cankers and the black-rot mummies, the mummified fruits should be destroyed and the cankers cut out where feasible. However, the chief reliance in most cases will have to be placed in fungicides, especially for control of fruit-rot and leaf-spot. Since black-rot cankers are mostly secondary and follow other primary injuries it follows that the chief attention in case of the cankers should be given to controlling those conditions which favor invasion by the black-rot fungus. For controlling the leaf-spot, the regular apple-scab spray schedule is said to be effective. For the fruit-rot, one or two applications of bordeaux, 4-4-50, about the middle of July and the first of August are recommended. Since the rot fungus invades the fruit largely through the codling moth tunnels, control of this insect will reduce black-rot infections.

#### LABORATORY STUDY OF BLACK-ROT

**A. Symptoms.** — Examine specimens illustrating the three forms of this disease, namely canker, fruit-rot, and leaf-spot.

1. *Canker.* — Observe cankers of different ages. What are the earliest symptoms of canker? On what aged cankers do the pycnidia appear? Are there any evidences that the black-rot fungus follows other injuries, that is, does this fungus seem to be a primary or a secondary pathogene? Draw typical cankers.

2. *Fruit-rot.* — Examine rotted fruits. What is the earliest evidence of rot? How does the color of the rotted area change as the rot spreads? When do the fruiting bodies of the fungus appear? What type of fruiting body does this fungus produce on the rotted surface? Compare with apple anthracnose and with apple-scab. Compare black-rot mummies with brown-rot mummies as to color, wrinkling, and presence of fruiting structures of the fungus on the surface. Draw.

3. *Leaf-spot.* — Examine leaves showing spotting caused by the black-rot fungus. Note early stage and later stages in the development of the spots. Describe the appearance. What is the basis for calling this the frog-eye leaf-spot? Does the fungus fruit on the leaf-spot? Draw.

**B. The fungus.** — In thin sections of diseased tissue or in pure cultures observe the vegetative mycelium of the fungus.

1. *Conidial stage.* — Examine sections of pycnidia from bark and from rotted fruit. Describe the pycnidium and the spores. Is there any variation in size, color and septation of the spores? Draw.

2. *Perfect stage.* — Where may the perithecia of this fungus be found? (See text.) If material is available note the characteristic habit of the perfect stage on the host and examine sections with the microscope. Compare with perithecia and ascospores of other fungi studied. Draw.

3. *Life cycle.* — How is the fungus perpetuated? How is dissemination brought about? What are the conditions for infection? When does canker infection occur?

Does fruit-rot attack young fruits or more nearly mature fruits? What part is played by the conidial stage? By the perfect stage?

C. Notes. — In the notes give attention to the geographic distribution of this disease; its economic importance in all phases, canker, rot and leaf-spot; symptoms; life history, giving attention to the question as to whether or not infection can occur in the absence of previous injury; and control measures.

#### REVIEW QUESTIONS

1. Give the known geographic distribution of the black-rot disease in the United States.
2. Describe the symptoms of this disease on fruit, stem and leaf.
3. Describe the morphology and life history of the causal fungus.
4. Is the black-rot canker considered a serious primary disease of fruit trees? Just what is its status in this respect?
5. Will the fungus attack apple fruits through the uninjured skin of the fruit?
6. What phase of the disease, canker, fruit-rot or leaf-spot, does the most damage, considering the country as a whole?
7. Discuss control measures.

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### Apple-scab

Caused by *Venturia inaequalis* (Cooke) Winter

Apple-scab is probably the best known and most widely distributed of all apple diseases. It has been commonly known under a variety of names in the different countries where it occurs so that the popular nomenclature of this apple disease is rather confusing. A list of common names applied to it includes such terms as black-spot, scurf, the fungus, and rust, in addition to the name "scab" by which designation it is usually known in the United States.



**Historical.** — The first mention of this disease seems to have been made by Fries (20) who reported it from Sweden in 1819. Wallroth reported it from Germany in 1833. It was apparently first reported from America by Schweinitz in 1834. The disease was first noticed in England in 1845, and in Australia (29) as early as 1862. Previous to 1866 only the imperfect or conidial stage of the scab fungus was known. This was given various names by the early mycologists but in 1869 Fuckel proposed the name *Fusicladium dendriticum* (Wallr.). This name is still used sometimes when speaking only of the conidial stage. In 1866 Cooke described the perfect stage as *Sphaerella inaequalis*, and in 1880 Winter (37) gave the ascigerous stage of the fungus its present name, *Venturia inaequalis*. It remained for Aderhold (1) to demonstrate the relationship of the *Venturia* stage to the *Fusicladium* stage of the fungus.

In Europe, during the latter half of the nineteenth century, Sorauer, Aderhold and others continued to publish series of articles on the apple-scab disease and its causal fungus, dealing with such phases of the subject as the symptoms on leaves and fruit, the botany of the fungus, life history studies, estimates of losses and methods of control. Aderhold (1, 2, 3, 4) during the years 1894 to 1900, published a long series of articles on the scab disease, in which he discussed such topics as the relation of the ascigerous stage, *Venturia*, to the *Fusicladium* stage, the time at which ascospores mature, varietal susceptibility, cultural and cross-inoculation experiments, and spraying experiments for control of the disease.

In the United States, during the last quarter of the nineteenth century and the first quarter of the twentieth century, a very extensive literature has grown up dealing with the life history of the scab fungus and more especially with experimental control. Life-history studies have been concerned largely with the manner of overwintering and the time and conditions of infection. Experiments on control have dealt largely with spray materials and spray schedules. From the time of the discovery of bordeaux mixture in 1883 until about 1908, this fungicide held first place as a spray for apple-scab. Since the introduction of the lime-sulfur sprays in apple-scab control by Cordley (13) in 1908, and by Scott (33) and others, this fungicide has largely replaced bordeaux mixture as a spray for the control of apple-scab. Still later the dusts, both sulfur and copper, have come into more or less competition with these liquid sprays in combating this disease.

**Geographical distribution.** — Apple-scab occurs to a greater or less extent in nearly all apple-growing sections throughout the world. It is found generally distributed over the United States but is much more

prevalent in the eastern and northern states, the Pacific Northwest, and the mountainous sections of Virginia and Arkansas. In some of the more southerly states it is of less importance than some of the other apple diseases such as bitter-rot and blotch. It is quite widely distributed in various countries of Europe and in Great Britain. The disease seems to be widespread and serious in Australia and New Zealand, and has been reported from South Africa.

**Hosts.** — The apple-scab fungus, *Venturia inaequalis*, seems to be confined in its attacks to species and varieties of apples. The pear-scab fungus is not the same, but a closely related species, while the peach, cherry and citrus-scab fungi are entirely different species.

There is some variation in the susceptibility of different apple varieties to scab but this difference in resistance does not seem to be constant. Schneiderhan and Fromme (32) list the following as susceptible: Wine-sap, Rome, Blacktwig, Ben Davis, Gano, Delicious, Stayman, Early Harvest; and as more resistant: Jonathan, Duchess, Transparent, York Imperial and Grimes. According to the Plant Disease Reporter, Supplement 28 : 280, in 1922 the order of susceptibility as reported for Vermont was: McIntosh, Northwestern Greening, Delicious, Northern Spy, Wolf River and Duchess; the first named dropping most of the leaves and setting no fruit because of scab, while the Duchess was practically immune. In Indiana the fruit of Grimes was badly scabbed in spite of the fact that this variety is usually considered practically immune. In Connecticut, scab was severe on McIntosh, Fall Pippin and crab apple; medium on Gravenstein; and slight on Red Astrachan, Delicious, Greening and Baldwin. According to Wallace (36) varietal susceptibility is not constant enough year after year to make possible a definite grouping of resistant and susceptible varieties. He states that certain varieties may be resistant in one year and susceptible in another year under similar conditions. It is probable, however, that the coincidence of favorable weather conditions with susceptible stages in the development of the host may account for this variation in scab occurrence.

**Economic importance.** — The loss occasioned by apple-scab when uncontrolled is undoubtedly enormous. Estimates of \$40 to \$50 per acre are not uncommon. The loss in one year in Illinois (34) was estimated at \$6,000,000. The damage caused by scab may be estimated by comparing the yield from blocks of sprayed trees with that from unsprayed trees. A survey of Niagara County, New York (17), indicated a gain of \$126 per acre during one season as a result of three applications of spray. As further evidence of the damage done by apple-scab it was estimated that in 1921 (5) the state of Washington suffered a loss of 2,673,000 bu. of apples from scab; Oregon, 1,032,000 bu.; New

York, 705,000 bu.; Illinois, 387,000 bu.; and other states smaller amounts. In 1922 (6) the reduction in yield in New York was placed at 13,091,000 bu.; Michigan, 2,257,000 bu.; and Pennsylvania, 5,386,000 bu. In 1923 (7) the loss in Kentucky was estimated at 1,676,000 bu. and in other states at smaller amounts.

*Nature of the loss.* — The damage done by this disease is by no means confined to the reduction in quality of scabby apples. While this source of loss is undoubtedly the most important item, there are other phases of the disease which sometimes cause a high percentage of loss. Blossoms are sometimes so severely attacked as to cause more or less reduction in set of fruit. The pedicels may be attacked and thus cause serious dropping of blossoms or young fruits. The foliage is subject to attack and while the disease may cause more or less defoliation the loss of leaves sufficient to cause serious injury to the tree is comparatively rare. Nevertheless the infection of only a few leaves early in the season results in a crop of spores which may cause serious secondary fruit infection later in the season.

*Symptoms.* — The symptoms of apple-scab are evident on the fruit, leaves, flowers and twigs.



FIG. 96. — Apple scab on the fruits.

*On the fruit.* — On the young, pubescent fruits scab is first manifest by a dark, olivaceous, sooty or smudgy appearance. Later after the pubescence has disappeared the spots are at first black or nearly black. As the spot enlarges the central, older part becomes brown and more or less corky in appearance, while the margin remains black. The cuticle becomes loosened around the margin of the scab thus giving it a whitish border (Fig. 96). Scab spots may finally be of any size up to covering practically one whole side of the fruit. In case of large scab spots the

corky surface of the spot may crack more or less. When new scab spots appear late in autumn, or in storage as sometimes happens, they differ somewhat from the early infections in that they are blacker in color and may get quite large before the brown corky appearance shows at the center of the spot. This latter stage may not be reached at all with the extremely late infections.

*On the leaves.*— The scab fungus may attack either surface of the leaf but frequently appears first on the lower side. The first noticeable effect is an olivaceous discoloration. At certain stages this discoloration may be described as a sooty or smudgy appearance. The spots vary in number and size and in extreme cases almost the whole surface of the leaf may be covered. The spots increase in intensity with age and finally the leaf tissue under the spot may die and turn brown. Dead, brown spots killed by scab can usually be distinguished from dead spots due to other causes by the fact that some of the dark, sooty growth, apparent in earlier stages, persists after the diseased spot has become brown and dead (Fig. 97).

*On the blossoms.*— Olivaceous, sooty spots similar to those occurring on the leaves may appear on the petals, sepals, receptacles or pedicels of the flowers (Fig. 98).

*On the twigs.*— It is stated that on some varieties and in some localities scab may appear on twigs. On the young, green shoots the spots are of a dark, sooty nature similar to infections on leaves and young fruits. As the twig grows older and becomes more woody the disease may cause rough, scurfy patches on the bark.

**Morphology of the fungus.**— The organism which causes apple-scab belongs to the class Ascomycetes and has a conidial stage as well as a perfect or ascigerous stage in its complete life history.

*Conidial stage.*— The conidial stage occurs on all affected parts, leaves, fruits, flowers and twigs. The conidiophores and conidia are densely effused, arising from a dense layer of mycelium which forms between the cuticle and epidermis. This mycelial layer is quite thin on the leaves and in the early stages does not penetrate beneath the epidermis. On the fruits the fungal layer is thicker and penetrates the epidermal cells to a greater extent. The conidiophores are unbranched and may become septate. The conidia are borne singly and terminally (Fig. 99). They are obclavate to lanceolate, at first unicellular but often becoming uniseptate, variable in size, measuring 12–22 by 6–9  $\mu$ . The conidia, conidiophores and mycelium are dark in color, ranging from olive to reddish-brown. In mass these give the scab spots a decidedly black appearance at times.

*Perfect stage.*— The perithecia occur on the dead leaves which have wintered on the ground. They appear as tiny black specks, barely

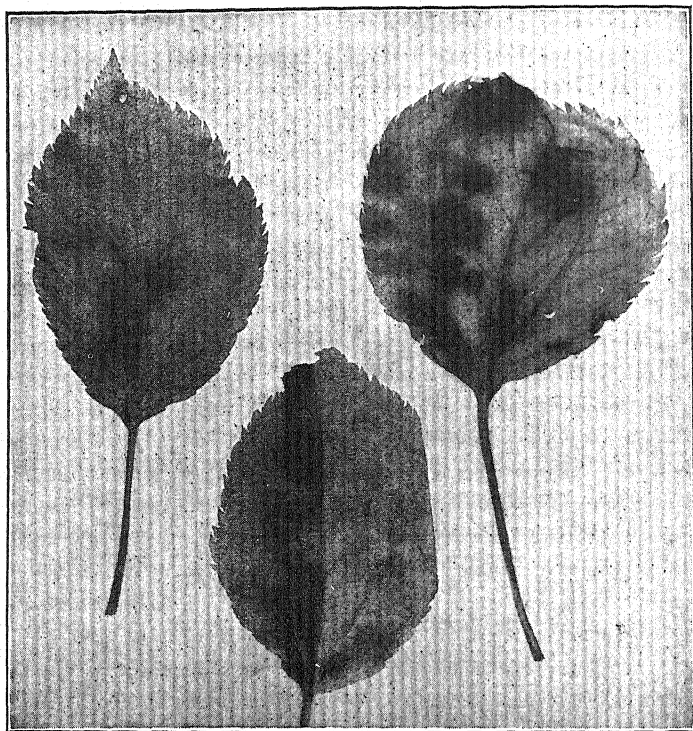


FIG. 97. — Apple-scab on the leaves.



FIG. 98. — Apple blossoms showing scab infection. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

large enough to be seen with the naked eye. The ostiolum of the perithecium emerges through the epidermis of the leaf. Each perithecium contains numerous asci each of which contains eight ascospores (Fig. 100). The spores are divided by a septum into two cells of unequal size, the shorter and broader cell always pointing upward as the spores lie in the ascus. Ascospores measure 11–15 by 4–8  $\mu$ .

**Life cycle.** — *Perpetuation.* — The apple-scab fungus probably over-winters almost entirely on the dead leaves on the ground. There is some evidence that it may possibly pass the winter as mycelium or conidia on the twigs (36), but this probably occurs rarely. When infected leaves fall to the ground in the autumn the mycelium continues to develop saprophytically in the dead leaf. Under favorable conditions the perithecia begin to develop probably soon after the leaves fall and by spring the ascospores are mature. The time at which the ascospores mature varies with climatic and weather conditions in different sections of the country but in any case they seem to be mature and in the process of dissemination at the time the leaves and flowers are unfolding in the spring. In Oregon (10) it has been demonstrated that ascospores may be ejected from the perithecia at any time from February to June. Experiments in Wisconsin (27) indicate that the time of leaf-fall in the autumn may bear a relationship to the time of ascospore maturity.

**Dissemination.** — The ascospores are forcibly ejected from the perithecia. Under the proper conditions of moisture the asci swell greatly and stretch lengthwise so that they protrude through the ostiole and beyond the surface of the leaf. Finally the ascospores are shot through an apical pore into the air where wind currents carry them to the new growth on the trees. There infection takes place and within about ten days to three weeks a crop of conidiospores matures. These conidia are in turn carried to other leaves and fruits chiefly by rain, and further infections occur, thus spreading the disease rapidly.

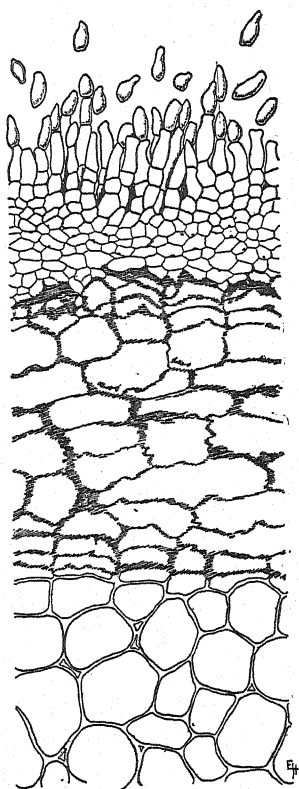


FIG. 99. — *Venturia inaequalis*. Section through a scab spot on apple fruit showing the conidial fruiting stage and several layers of cells of the apple pulp beneath the epidermis.

**Infection.** — Both ascospores and conidia are able to infect blossoms, leaves, fruits, and young twigs, whenever the proper conditions prevail. Considerable moisture, in the form of rain or heavy fogs, is a requisite

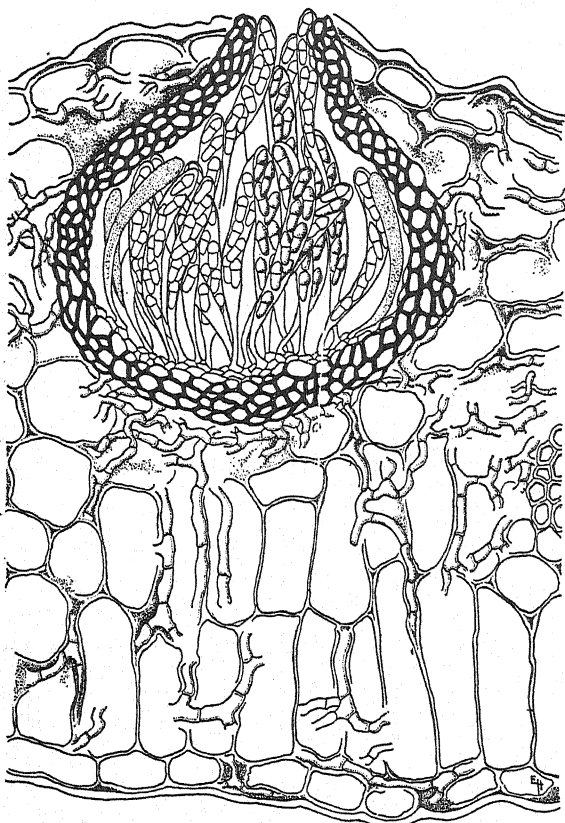


FIG. 100. — *Venturia inaequalis*. Section through a perithecium imbedded in a dead leaf, showing the asci and ascospores.

for spore germination and infection. In general, cool, wet weather is conducive to the germination of the spores and infection of the host. On the other hand, hot, dry weather is unfavorable for the spread of the disease. If very dry weather prevails for a few weeks during the time of blossoming and unfolding of the leaves there is little opportunity for the primary ascospore infections to occur, hence there will be few conidia for secondary infection.

**Control.** — In discussing control of the apple-scab fungus two life history facts must be kept in mind. (a) The

primary infections are initiated by ascospores coming from the dead leaves and infecting the young leaves, blossoms and fruits. (b) Secondary infections are brought about by conidia which result from the first ascospore infections.

**Sanitation.** — Control measures, then, will necessarily be based upon the above facts. Any measures which will reduce the number of dead leaves or of ascospores coming from them and lodging on the green leaves, blossoms or young fruits will be useful items in control. Burning or plowing under the dead leaves will reduce the number of spores.



Spraying the leaves on the ground has been suggested (6) but is ineffective because the ascospores are protected in the heavy-walled perithecium and upon escaping are shot out into the air beyond reach of any fungicide on the surface of the leaf. Cover crops are useful because they grow up over the dead leaves and prevent air currents from catching up so many of the ascospores and bearing them to the tree.

*Spraying.* — If every dead leaf in the whole community could be destroyed before any ascospores escape, the problem of control would

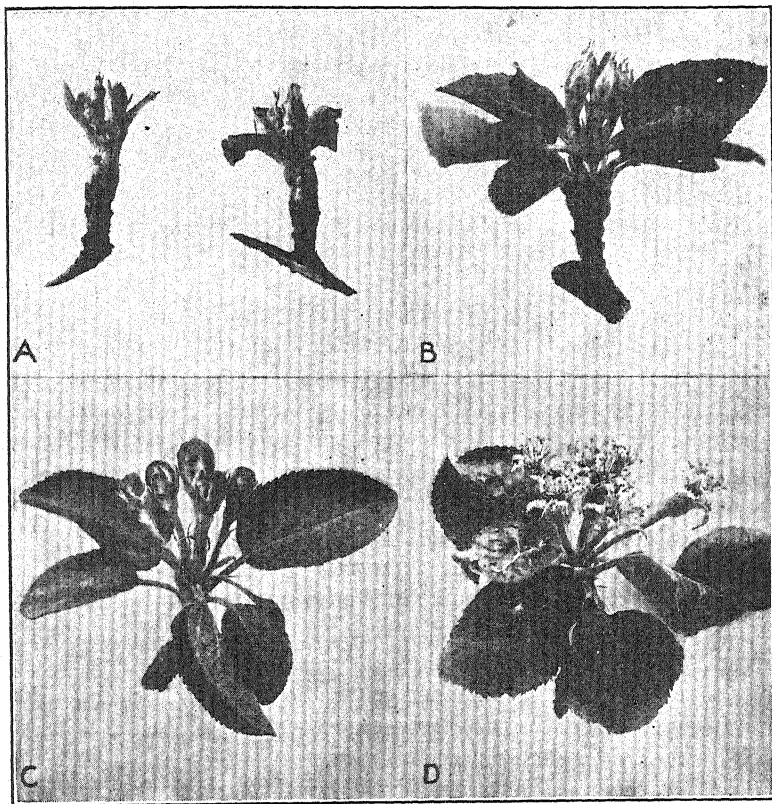


FIG. 101. — Apple spurs showing the various stages in the development of leaves and blossoms which are suggested in scab spray schedules as the proper intervals for the application of the first four sprays. A, delayed dormant; B, pre-pink; C, pink; D, calyx. (D, from photograph furnished by Department of Entomology, Ore. Agr. College.)

be largely solved, except in case the fungus might live over on the twigs, which apparently is not an important item. But since it is practically impossible to destroy all the leaves it becomes necessary to use other



means of control. Spraying for apple-scab is based upon the principle of protecting all susceptible parts against infection by either ascospores or conidiospores. Since ascospores are present before, during and after the buds open and conidiospores may be present all summer, provided all early infections are not prevented, it becomes necessary to use a very comprehensive spray program for this disease. The following spray schedule is recommended in localities where apple-scab is serious.

(1) *Delayed dormant spray*: Applied as soon as the leaves begin to emerge from the bud. (Fig. 101 A.)

(2) *Pre-pink spray*: Applied when the cluster of flower buds has emerged so as to show the individual buds but before the calyx has opened enough to show the color of the petals. (Fig. 101 B.)

(3) *Pink spray*: Applied when the individual flower buds have expanded enough to show the color of the petals but before the blossoms are in full bloom. (Fig. 101 C.)

(4) *Calyx spray*: Applied just after most of the petals have fallen but before the calyx lobes have closed over the blossom end of the fruit. (Fig. 101 D.)

(5) *Ten-day spray*: Applied ten days after the calyx spray.

(6) *Thirty-day spray*: Applied thirty days after the calyx spray.

Whether or not it will be necessary to carry out all of the above schedule depends upon conditions in any particular case. If scab is present to any extent, the first spray cannot safely be omitted because it has been shown above that ascospores are apt to be present at the time the buds begin to open. If the first three sprays have been used so effectively that no infections have occurred and especially if the weather is dry, the latter applications may be omitted with safety. On the other hand if some early infections have occurred and if the weather remains sufficiently humid for infections to take place later in the summer, the number of applications may have to be increased considerably above the five recommended in the schedule. It cannot be too strongly emphasized that the grower must study the conditions with which he has to deal and act accordingly.

*Spray materials.* — Both lime-sulfur and bordeaux mixture are effective against scab, and the dusts have been used with more or less success. In the early season lime-sulfur is preferable because of the tendency of bordeaux to russet the young fruit in cool weather. During hot weather lime-sulfur sometimes causes burning and in the latter case it may sometimes be advisable to change to bordeaux for the later applications.

*Thoroughness essential.* — The one point that should be emphasized perhaps above all others is thoroughness. Every bit of leaf and fruit

surface must be covered with spray in order to protect it adequately. Childs (9) calls attention to the difficulty of doing a good job of spraying under adverse conditions such as tall trees and high winds. He shows that under average conditions the fruits in the top of a tall tree are not as thoroughly protected as those on the lower limbs unless special precautions are taken.

#### LABORATORY STUDY OF APPLE-SCAB

**A. Symptoms.** — Make a careful study of the symptoms of apple-scab as exhibited on the fruits, leaves, blossoms and twigs.

1. *On fruits.* — Examine fruits of different ages showing different stages in the development of scab infections. Compare early symptoms with older infections. How deep does the lesion extend at first? Later? Note the olivaceous, smudgy appearance of the early stages and the rough, corky nature of the older scabs. Note how the cuticle is lifted and peels off around the edges of the older lesions. **Draw.**

2. *On leaves.* — What are the first signs of the disease on leaves? Later symptoms? Does the fungus ever kill any of the leaf tissue? Examine dead apple leaves picked up from the ground in the spring and containing the scab fungus. What signs are observable? Of what does the dark, smudgy growth found on newly infected leaves consist? **Draw.**

3. *On blossoms.* — Compare infected blossoms with diseased leaves in all details. What flower parts are liable to attack by the scab fungus? What harm may result from flower infection? **Draw.**

4. *On twigs.* — Is twig infection as common as leaf and fruit infection? What aged twigs are susceptible? What are the symptoms on twigs? Compare with symptoms on other parts. **Draw.**

**B. The fungus.** — Both conidial and perfect stages occur in the life cycle.

1. *Vegetative mycelium.* — Examine sections of fruits and of green leaves showing the conidial stage. Note that the mycelium is largely confined to the region between the cuticle and the epidermis. On the fruit-scab spot observe the dense layer of fungous tissue between the cuticle and the epidermis. The cuticle may have disappeared over a large part of the scab spot but may be seen around the margin of the lesion.

2. *Conidial stage.* — These spores are found on the surface of scab spots wherever they occur, on fruit, leaf, flower, or twig. Make mounts by scraping off some of the spores with a scalpel, or better, use prepared sections cut through scab spots from fruit or leaf. In the sections note the continuous layer of conidiophores and conidia. **Draw.**

3. *Perfect stage.* — Study sections cut through the perithecia on the old dead leaves. Note particularly the shape, septation and arrangement of ascospores within the ascus. **Draw** section of perithecium. **Draw** one ascus with its spores, much enlarged.

4. *Life cycle.* — Where does the fungus overwinter? How is it disseminated? When, where and under what conditions do the initial infections in the spring occur? What spores are responsible for the first infections? How does the fungus spread later in the season?

5. *Cultures.* — Cultures of this fungus can be made easily in the spring at the time ascospores are discharging from the dead leaves. Suspend a piece of leaf containing perithecia over an agar plate. Ascospores will be discharged on to the agar where

they will germinate and form colonies of the fungus. Then make transfers to agar tubes. With proper precautions pure cultures can be secured in this way.

C. Field study. — If season and conditions permit, this disease may be studied profitably in the field. The following points are of interest.

1. Find the perfect stage on old leaves.
2. Determine the seasonal range of ascospore discharge.
3. Observe new infections on flowers, leaves and young fruits.
4. Determine the time necessary to produce a crop of conidia after inoculation.
5. Estimate percentages of infection.
6. Watch the development of fruit and leaf lesions throughout the season.

D. Notes. — Follow the outline on page 152 and write complete notes on this disease. Give particular attention to method of overwintering, and time and place of infection, with reference to the bearing these factors have on control.

#### REVIEW QUESTIONS AND PROBLEMS

1. Describe the symptoms of apple-scab on all parts of the host.
2. Give the complete life-history story of the scab fungus, paying particular attention to perpetuation, dissemination and infection. (See reference 36.)
3. Discuss apple-scab epiphytotics in relation to weather conditions. (See reference 28.)
4. Discuss the question of regional variations in the severity of apple-scab and the consequent variation of control practices for various sections of the country.
5. Discuss the relation of height of fruit on the tree to apple-scab infections. (See reference 9.)
6. Why will not dormant sprays control apple-scab?
7. Compare the efficacy of dusting versus spraying for the control of apple-scab. (See references 23 and 31.)
8. What is the source of inoculum for the primary infections of apple-scab? The secondary infections?
9. Give a full report on the economic importance of apple-scab. (See references 5, 6, 7, 34 and 36.)
10. Discuss the question of resistant and susceptible varieties of apples. (See references 32 and 36.)

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### Pear-scab

Caused by *Venturia pyrina* Aderh.

Pear-scab is caused by a species of *Venturia* very closely related to the apple-scab fungus. The symptoms of the disease, the life history of the causal fungus, and the control measures are all very similar to those of apple-scab. The most striking morphological difference in the two species of fungi is found in the ascospores. In both species the ascospores are once septate, consisting of one longer and one shorter cell. In *Venturia inaequalis*, the apple-scab fungus, the shorter of the two cells composing each ascospore is found at the uppermost end of the spore as it lies in the ascus, while in *Venturia pyrina*, the pear-scab fungus, the shorter cell is at the lower end of the spore. The two species are distinct physiologically as well as morphologically, pears being immune to the apple-scab fungus, while the pear-scab fungus is likewise unable to attack apples.

#### LABORATORY STUDY OF PEAR-SCAB

1. Compare the symptoms of pear-scab in all details with those of apple-scab. Are fruits, leaves, blossoms and twigs attacked as is the case with apple-scab? Are the symptoms on all these parts comparable in the two diseases? If there are any differences, point them out.
2. Compare the two fungi in all details of both conidial and perfect stages. Point out any differences that exist. Compare their manner of overwintering and dissemination.

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### Blight or Endothia Canker of Chestnut

Caused by *Endothia parasitica* (Murr.) And. & And.

Near the beginning of the present century there was discovered in New York a disease of the chestnut tree hitherto unknown on the North American continent. The malady proved to be a most serious bark disease of this valuable forest tree, spreading very rapidly, and soon threatened to exterminate the chestnut throughout its range in the eastern United States. Various names have been applied to this disease, among which are chestnut-blight, chestnut canker, chestnut bark disease and Endothia canker. The last-named term seems to be a desirable name by which to designate this disease.

**History and distribution.** — The Endothia canker of chestnut was first discovered by Merkel in one of the parks of New York City in 1904 and reported by him two years later (13). The disease spread rapidly and was soon recognized as a menace to the chestnut forests of the eastern United States, hence it immediately became the object of intensive study by many plant pathologists and mycologists. By 1908 the disease was reported as serious in portions of Pennsylvania, New Jersey, New York, Long Island, Connecticut and Massachusetts. In 1914 the range of the disease had extended so that it was generally prevalent from southern Vermont, New Hampshire and eastern New York southward into northern Virginia and westward to central Pennsylvania. Outside of this area scattering infections were found in Maine, western Pennsylvania and North Carolina. The origin of the disease was in doubt until in 1913 when it was discovered by Meyer in China (6). It then became apparent that the fungus probably had been introduced into the United States on chestnut nursery stock imported from the Orient for ornamental purposes. This theory was strengthened by the discovery of the disease in Japan in 1915.

In Pennsylvania the threat of the disease to exterminate the valuable stands of chestnut led to the appropriation of a large sum of money and the establishment of a Commission to investigate the trouble and to devise means by which it could be controlled. The Federal Government also made provision for investigating the disease and for several years intensive work was done on the problem by both state and federal agencies.

**Hosts and varietal susceptibility.** — The fungus, *Endothia parasitica*, has been reported on a number of species of chestnut as well as on several other species of forest trees. The Japanese and Chinese species of chestnut are so resistant to the canker fungus that little injury results from its attacks. On the other hand the American chestnut, *Castanea*

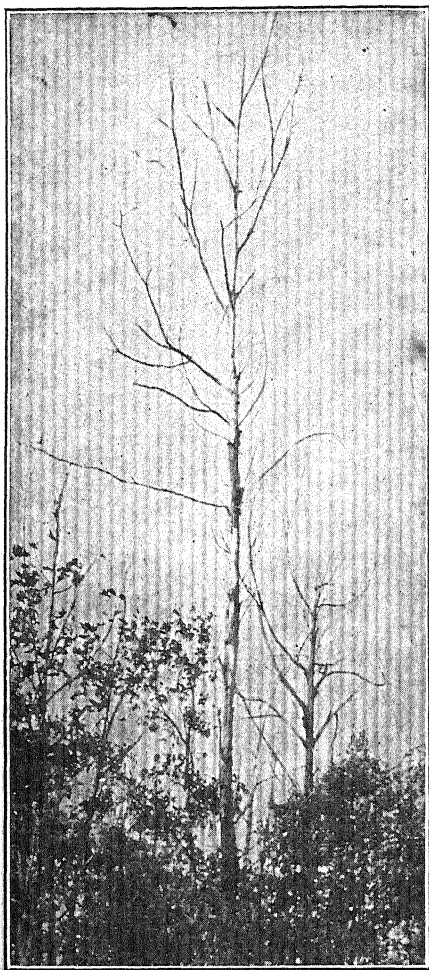


FIG. 102. — Chestnut trees killed by *Endothia* canker. (Cornell Univ. Agr. Exp. Sta. Bul. 347.)

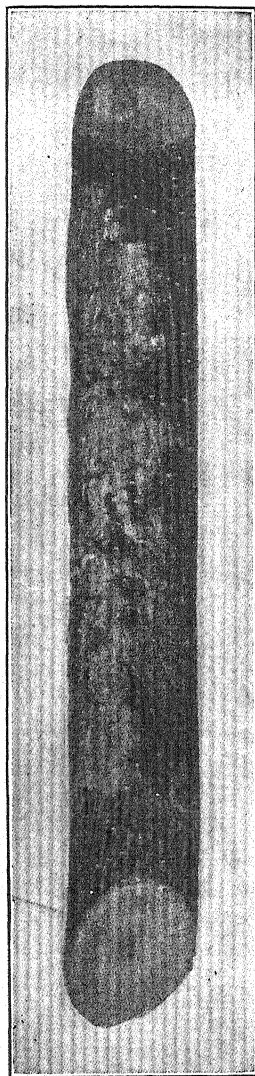


FIG. 103. — Chestnut canker. Ameboid infection on two-year-old shoot. (After Heald.)

*dentata*, is very susceptible and soon succumbs to the disease. This is a good illustration of an imported parasite finding a more susceptible host in its new habitat. Evidently this fungus has lived on the chestnut trees in Asia for ages without materially damaging them but upon being



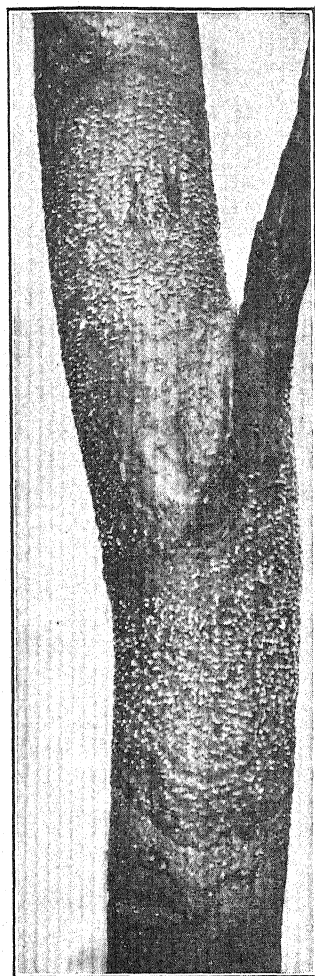


FIG. 104. — Chestnut canker. Characteristic pycnidial fruiting pustules covering the dead area. (Cornell Univ. Agr. Exp. Sta. Bul. 347.)

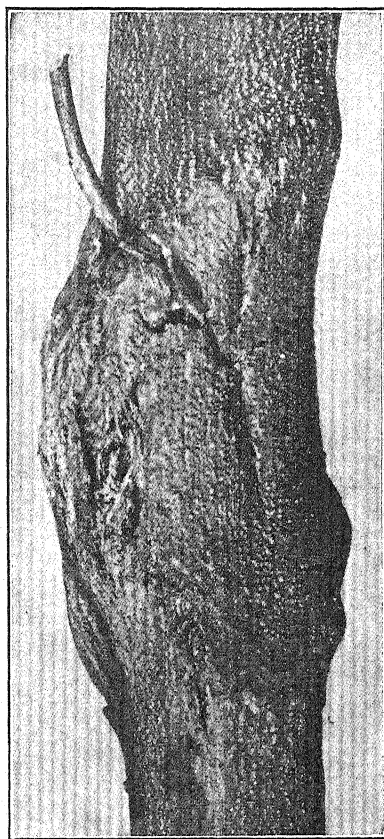


FIG. 105. — Chestnut canker. The swollen type of lesion. (After Heald.)

introduced to our American species at once becomes a menace to its existence (see Chapter IX). Both the eastern Chinquapin, *Castanea pumila*, and the western Chinquapin, *Castanopsis chrysophylla*, while not immune, are much more resistant than the American chestnut. The European chestnut, *Castanea sativa*, also is susceptible. The fungus has been found growing saprophytically on dead bark of various other trees but apparently is not able to attack living trees of genera other



than *Castanea* to a serious extent. Inoculations on various species show that most of them are immune. The fungus was able to grow slightly in one or two species of oak and two trees of *Rhus* were killed (20).

**Economic importance.** — The chestnut is, or was, one of the main forest trees of Connecticut, New York, Pennsylvania and southward in the Allegheny mountains to Alabama. Some idea of its value may be gained by noting the value of cut timber for several years before the *Endothia* fungus had wrought its destruction. In 1907 the total cut of chestnut for all purposes including lumber, posts, poles, rails, cross-ties and tan-bark, was valued at \$19,188,219. In 1909 the value of all chestnut timber products was again estimated at approximately the same figure (3). This tree is also highly prized as an ornamental and has been used extensively in parks and large estates. When one reflects that the canker disease has practically exterminated this species over

most of its natural range the great economic loss inflicted by its ravages can be readily appreciated.

**Symptoms.** — The symptoms of this disease may be listed in general under two heads: (a) the canker effects; and (b) the effects on the parts beyond the cankers. On young shoots typical cankers are formed, which may first be recognized as yellowish or yellowish-brown patches and may be regular or irregular in outline. The diseased area may be either nearly circular or elongated in a longitudinal direction (Fig. 103). On older branches or trunks the first symptoms appear as slightly discolored dead areas. On old fissured bark little change in the appearance of the bark can be noted until the infections are quite old. The cankers usually continue to enlarge until girdling results.



FIG. 106. — Chestnut canker. Old lesion on branch that has been dead for a year. (After Heald.)

Sometimes a marked enlargement or hypertrophy results, involving all or a part of the cankered area (Fig. 105). On old cankers the bark becomes rough and much cracked (Fig. 106). As the invasion of the fun-

gus progresses the mycelium spreads out in the bark and may be observed in whitish or buff-colored mats if the outer cork layer of bark is peeled off, or later these mycelial fans may appear in the cambium zone. As the development of the canker proceeds the pycnidial fruiting

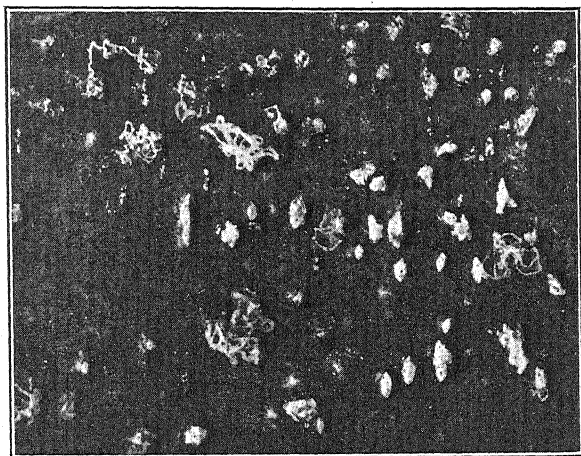


FIG. 107. — Chestnut canker. Characteristic tendril-like spore horns extruded from the pycnidia. (Photograph from Cornell Univ. Agr. Exp. Sta.)

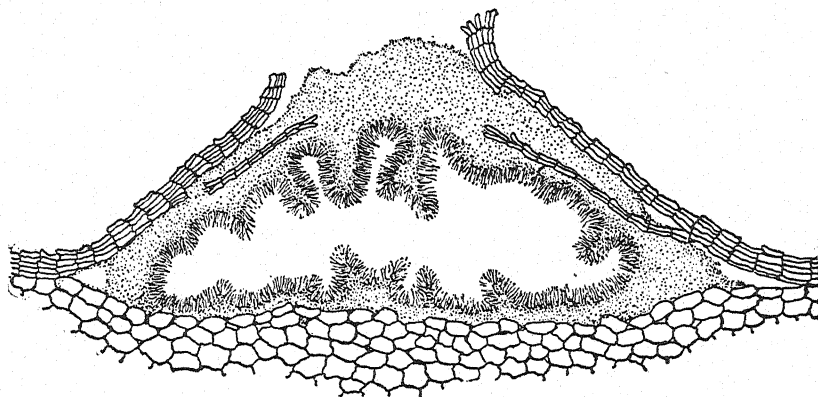


FIG. 108. — Section of pycnidium of *Endothia parasitica*. (After Heald.)

pustules break through the surface of the bark (Fig. 104). When moist conditions prevail the pycnospores ooze from these pustules in long twisted tendril-like threads or "spore-horns" (Fig. 107). Later the fungus gives rise to the perfect stage fruiting bodies, the stromata containing perithecia, which are larger and more conspicuous than the pycnidia (Fig. 110).

When twigs, branches or trunks are girdled the distal parts die.

Various symptoms appear following or in conjunction with this girdling. On some dead twigs, clusters of dead leaves may remain all winter. Burs also frequently cling persistently to dead twigs. Leaves may become chlorotic or brown and finally fall leaving bare twigs or branches. When many branches on a tree have been killed a "stag-head" effect results.

**Morphology and life history of the fungus.** — The characteristic fans of the vegetative mycelium have been mentioned above under Symptoms. The fruiting structures consist of a pycnidial stage and a perithecial stage. The pycnidia may develop at any season of the year depending upon the age of the lesion and the temperature and moisture conditions. On smooth-barked young cankers, the pycnidia appear as somewhat globose bodies which develop under the outer cork layer and push it up in numerous small blisters. The outer bark is ruptured and through an opening or ostiole the

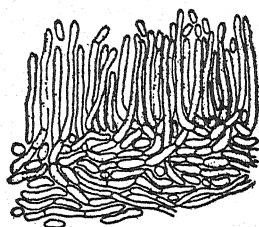


FIG. 109. — Section of wall of a young pycnidium showing conidiophores and conidia. (Cornell Univ. Agr. Exp. Sta. Bul. 347.)

tendrils of spores emerge. The cavity of the pycnidium is about a fourth of a millimeter in diameter and at first is almost round in cross section but becomes irregular with age (Fig. 108). The walls of the cavity are lined with conidiophores from which the conidiospores are cut off successively (Fig. 109). These spores are oblong or cylindrical in shape, have rounded ends and measure about  $1.28$  by  $3.56 \mu$  in size.

Later development of the pycnidia may transform them into peri-

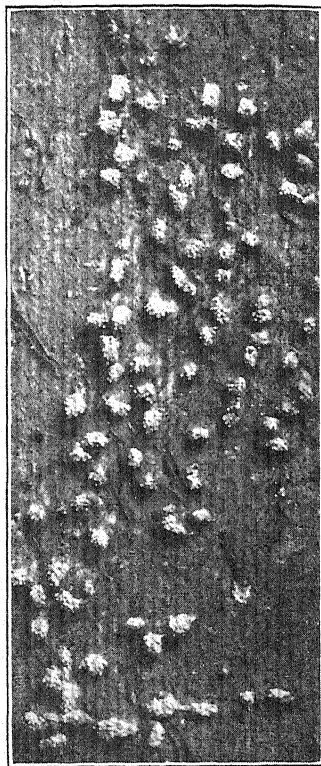


FIG. 110. — Stromata of *Endothia parasitica*. (Cornell Univ. Agr. Exp. Sta. Bul. 347.)

thecial stromata. As the stroma enlarges there are formed within it a number of flask-like cavities with long necks which open to the surface through minute ostioles which show on the surface as raised papillae or minute black dots. These flask-shaped cavities are perithecia (Fig. 111), each of which contains a large number of eight-spored asci (Fig. 112). The asci are broadly clavate or oblong and average  $51.2$  by  $8.9 \mu$ . The ascospores are oblong to oval in shape, two-celled, constricted at the septum, and average about  $4.5$  by  $8.6 \mu$  in size (3).

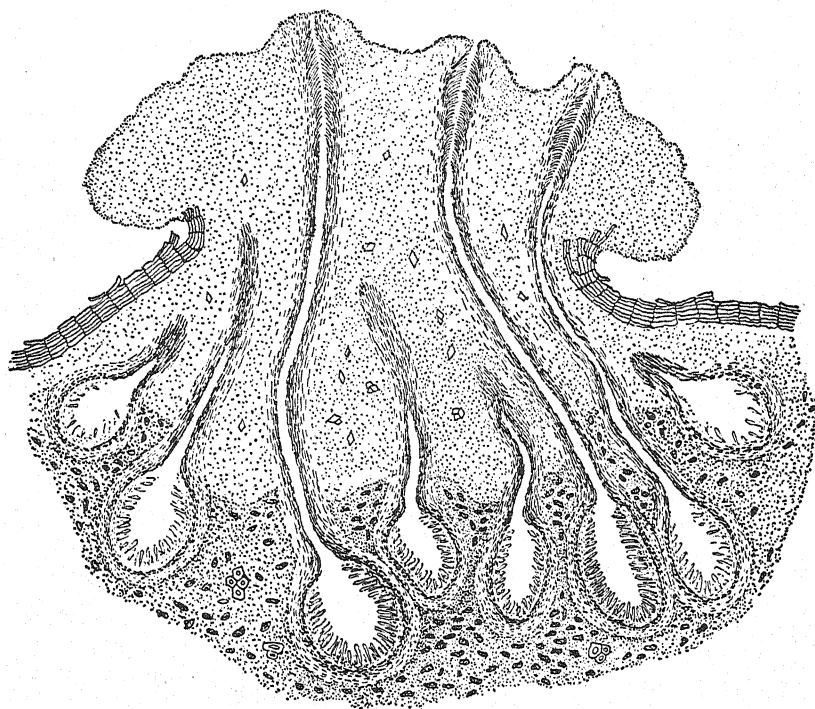


FIG. 111. — Section of a stroma of *Endothia parasitica* showing the long-necked perithecia. (After Heald.)

The fungus is perpetuated in the cankers and both pycnospores and ascospores may be produced at any season of the year. Either form is capable of producing infection. Pycnospores are disseminated largely by rain, insects and birds, while ascospores are wind-disseminated. Infection takes place only through some injury to the bark.

**Control.** — Several years of intensive study by competent plant pathologists in the employ of the Pennsylvania Chestnut Tree Blight Commission, the United States Department of Agriculture and several

state experiment stations failed to solve the problem of controlling this disease. In spite of all the efforts put forth the malady has continued to spread steadily and now all hope of saving the native stands of commercial chestnut in the United States has been abandoned. It is true

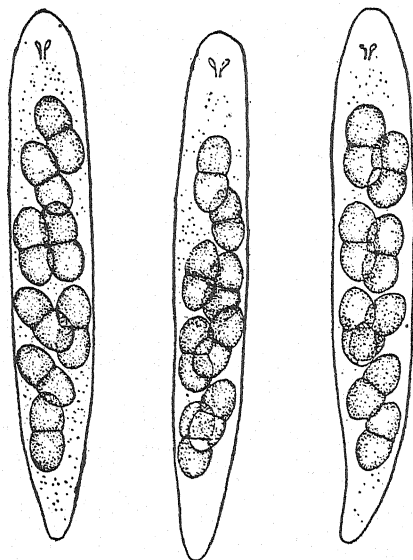


FIG. 112. — Asci and ascospores of *Endothia parasitica*. (After Heald.)

that certain canker-resistant individual chestnut trees have been found and it may be possible to breed resistant varieties for future plantings but for the present the only recourse seems to be the substitution of other species of timber trees in those areas formerly largely occupied by the American chestnut. As for isolated orchards or plantings it may be possible to save them by the intensive cutting out procedure now used in combating fire-blight in pears. This will be possible, however, only in case of complete isolation, otherwise the disease will come in again as rapidly as it is cut out.

#### LABORATORY STUDY OF ENDOTHIA CANKER

**A. Symptoms.** — Examine all available specimens showing the various symptoms and signs of *Endothia* canker. Observe early and late stages of the canker on both young twigs and older branches. Note the color of the bark, both on the surface and under the surface. Is the margin of the canker distinctly marked? Is there either hypertrophy or shrinkage of the affected parts? Do you find any specimens which show splitting or cracking of the affected bark? Is the fungus fruiting on the diseased area? Can you distinguish pycnidia from perithecia? Look for the mycelial fan under the cork layer or deeper in the bark. You may find it in the cambium region. Examine specimens or look at illustrations showing the blighting effect on twigs and branches resulting from girdling. Make drawings to illustrate the symptoms observed.

**B. The fungus.** — Examine sections of both pycnidia and perithecia and note the spores and the spore-bearing structures of each. Draw section of both pycnidium and perithecium. Draw conidiophore and conidium, also an ascus containing ascospores, much enlarged.

Look up the life history of the fungus and learn the method of perpetuation, dissemination and infection, and the conditions under which these processes take place.

**C. Notes.** — Write notes including particularly such items as history, distribution, symptoms, life history and control.

## REVIEW QUESTIONS

1. What is probably the native home of the chestnut-canker fungus?
2. How is it supposed to have been introduced into this country?
3. What species of trees are in any degree susceptible to this disease? Which species of chestnut is most susceptible? Which most resistant?
4. How does this disease illustrate the disturbance of the "biological equilibrium" as discussed in Chapter IX?
5. What is the range of the American chestnut? What are its economic uses?
6. Describe the symptoms of the Endothia canker disease.
7. Describe the morphology and life history of the causal fungus.
8. Discuss the efforts made to combat this disease and the degree of success attained.
9. How do you account for the difficulty met in combating this disease?
10. Is there any probability of developing resistant strains of chestnuts? (Reference 9.)
11. Discuss the disseminating agents of the chestnut canker fungus. (References 2, 12, 21.)

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ADDITIONAL LIST OF DISEASES CAUSED BY ASCOMYCETOUS FUNGI, WITH  
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## CHAPTER XIX

### DISEASES CAUSED BY FUNGI — BASIDIOMYCETES

The Basidiomycetes are distinguished from the other classes of fungi by the presence of the basidium, a more or less club-shaped structure, sometimes unicellular and in other cases septate, on which the principal spores, basidiospores, are borne. The fungi of this class are also characterized by septate mycelium, a character which separates them from the Phycomycetes but not from the Ascomycetes nor from the Fungi-Imperfecti, a class to be discussed later. There are several thousand species of Basidiomycetes, not all of which are of importance from the standpoint of plant pathology. However, there are several groups of this class of fungi which rank well up with the Ascomycetes and the Phycomycetes as serious plant pathogens. The three subgroups of the Basidiomycetes which are of greatest importance to the plant pathologist are represented respectively by (a) the smuts (Ustilaginales), (b) the rusts (Uredinales), and (c) the mushrooms and bracket fungi (Agaricales). Of these three orders the mushrooms and bracket fungi are the only ones possessing the typical basidium as illustrated in Fig. 150 B. In the rusts the basidium appears in a modified form known as a promycelium (Fig. 130). In the smuts the basidium is still more extensively modified but is also termed a promycelium (Figs. 115, 123).

#### The Smuts

The smuts caused by fungi belonging to the order Ustilaginales are among the most destructive of plant diseases. The most important smut diseases are those occurring on the cereals, but many other plants including the sorghums, millet, timothy, and the onion are also severely attacked by different species of the smut fungi. The various grain crops are susceptible to a number of different smut-producing species of fungi, each of which usually attacks only a particular kind of cereal. The important smuts found on the common grain crops, wheat, oats, barley, rye and corn, may be classified in three groups depending upon the life history of the causal fungus, especially as to the time, place and method of infection. The following outline shows the group to which each species of smut belongs.

## I. Seedling infection group.

1. Stinking smut or bunt of wheat caused by *Tilletia tritici* and *T. laevis*. (Fig. 113.)
2. Loose smut of oats caused by *Ustilago avenae*. (Fig. 116 A.)
3. Covered smut of oats caused by *Ustilago laevis*. (Fig. 116 B.)
4. Covered smut of barley caused by *Ustilago hordei*. (Fig. 117.)
5. Flag smut of wheat caused by *Urocystis tritici*.
6. Rye smut caused by *Urocystis occulta*.
7. Head smut of corn caused by *Sphacelotheca reiliana*.

## II. Blossom infection group.

1. Loose smut of wheat caused by *Ustilago tritici*. (Fig. 118.)
  2. Loose smut of barley caused by *Ustilago nuda*. (Fig. 120.)
- This smut belongs in both Groups I and II.

## III. Local infection group.

1. Common smut of corn caused by *Ustilago zaeae*. (Fig. 121.)

It should be remembered that the above classification has nothing to do with the symptoms exhibited by the different smuts nor with any other phenomenon except the manner of infection. In Group I infection occurs in the young seedling shortly after the seed has germinated and usually before the seedling has emerged from the ground. In this type of smut the spores are mature and disseminated at harvest or threshing time and contaminate the sound seed. In some cases also the soil is contaminated. For this type of infection the smut spores must either be carried on the seed or be present in the soil in close proximity to the germinating seed. In Group II, infection occurs in the ovary of the flower at blossoming time. In this type the smutted heads are mature at blossoming time and the spores are disseminated by the wind and lodge within the flower. In Group III, infection may occur on any of the tender growing parts of the plant. Leaves, stems, ears and tassels of the corn plant may become infected locally from spores which lodge upon these parts while they are yet in a growing and susceptible condition. In this case the spores must winter over in the field and be disseminated during the next growing season. In the following pages one smut from each group will be discussed in detail. The symptoms of some of the others are illustrated and references on the smuts which are not discussed in detail are appended. From Group I, the stinking smut has been selected for full discussion but control measures for the other seedling infection smuts are appended where these measures differ from those recommended for bunt.

### Bunt or Stinking Smut of Wheat

Caused by *Tilletia tritici* (Bjerk.) Wint. and *T. laevis* Kühn

It is entirely probable that this smut as well as other species of grain smuts was known in ancient times to those who cultivated the cereal crops. However, owing to the confusion of terms it is difficult or impossible to identify the species in ancient writings. Both the Greeks and Romans seem to have used the same term to apply to both rusts and smuts. In early English writings "mildew" was used as synonymous with smut. In view of the entire lack of knowledge of the nature of fungi then existing it is not surprising that no sharp distinctions were made between smuts, rusts and mildews.

Apparently Tillet was the first to establish the infective nature of the bunt dust. He did this, in 1755, by sowing smutted seed alongside of rows of clean seed and found that the smutted seed produced a smutty crop and the clean seed produced smut-free wheat. Even then he did not recognize the true parasitic nature of the bunt fungus. Prevost (24), in 1807, described the germination of the spores and the production of sporidia. Kühn (20), in 1859, observed the entrance of fungus threads into the host plant and established the fact of seedling infection by this smut fungus.

**Distribution.** — Apparently the stinking smut of wheat occurs wherever wheat is grown all over the world. Its severity varies greatly, however, in different localities. In some regions it is of less importance than the loose smut while in other places it takes first rank among cereal diseases, causing enormous loss in bad smut seasons. In the United States the greatest damage resulting from bunt probably occurs in the wheat-growing sections of the Pacific Northwest, especially in eastern Washington and Oregon, and in Idaho. Wheat-growing states east of the Rocky Mountains also suffer heavy losses during some seasons.

**Losses.** — The damage done by bunt fluctuates from season to season and in various localities. Losses are attributable to two separate and distinct items, namely, actual percentage of decrease in yield, and dockage at the market on smutted grain. In Kansas reports of loss from stinking smut range from 1 to 20 per cent (22). The states of Washington, Oregon and Idaho are estimated (14) to suffer an average annual loss of 4,000,000 bu. of wheat due to bunt. In 1923 the total estimated (2) loss for the entire United States was 1.27 per cent or 11,308,000 bu. In that year the total loss in the state of Illinois was estimated at \$2,375,000. Of this amount the cash dockage on smutted wheat amounted to \$100,000 while the remainder was due to decreased

yield. In 1924 the total loss for the United States (4) was 2.72 per cent or 26,038,000 bu. This loss was distributed in many states, ranging from 8 per cent in Kansas and Idaho to .5 per cent in Texas, Nebraska and Pennsylvania, a trace in Kentucky and New York, and none in Connecticut.

During the three years immediately preceding April 1, 1926, it is estimated (6) that over 128,000 carloads of wheat arriving at the terminal markets in the United States were graded as "smutty." Smutty wheat must be cleaned before it can be used for flour. The scouring process is expensive so that smutty wheat suffers considerable dockage, usually ranging from a few cents to as much as 20 cents per bu. During the period from July 1, 1925, to March 31, 1926, 42.1 per cent of the wheat arriving at Omaha was graded smutty, at Portland, Oregon, 29.6 per cent, and at Duluth, 17.4 per cent.

The Plant Disease Reporter, Supplement 53, 1927, states that during the years 1924, 1925 and 1926, stinking smut caused greater loss in the United States than any other wheat disease. It is also stated that in 1926, stinking smut caused the greatest loss ever recorded for this disease. Data taken at various terminal markets indicate that, for example, of 22,000 cars of wheat received at the Kansas City market during August, September, October and November, 1926, 25 per cent graded smutty. Discounts on these smutty cars averaged approximately \$49 per car or a total of \$272,360.

In addition to losses due directly to smut and to dockage, another kind of loss has to be considered. In sections where smut is abundant a large number of explosions and fires occur in separators during the threshing season. In the state of Washington during the summer of 1914 it was estimated that about 300 fires or so-called explosions occurred (10). These fires were presumably due to the ignition of the smut dust. In many of these fires the threshing machines were destroyed and in some cases further loss was sustained due to the burning of wheat.

**Hosts.** — The chief crop attacked by the stinking smut fungi is wheat. The wheat varieties, emmer, spelt and einkorn are also somewhat susceptible. Bunt is known to occur occasionally also on rye.

The various strains and varieties of wheat show a marked difference in susceptibility to bunt. According to Tisdale and others (27), nearly all varieties of American wheats, all of the Australian wheats except one, and all of the South African and Indian wheats are more or less susceptible to stinking smut. The hard red winter wheats are the least susceptible of the four commercial classes of common wheat. The white wheats are the least resistant as a class but this group contains a

few very resistant strains which are an exception to the rule. Most of the soft red winter and hard red spring varieties are more or less susceptible but one variety of soft red winter, Banner Berkeley, proved very resistant. The Club wheats as a rule are quite susceptible to stinking smut. Two varieties, Hussar (C. I. 4843) and Martin (C. I. 4463), are entirely immune to bunt. Certain strains of White Odessa and Turkey, and also Ridit, Florence, and Banner Berkeley varieties show great resistance. Most of these immune or resistant strains are not of high commercial value but are being used in breeding experiments with good prospects of developing strains which will be both commercially desirable and immune to smut. A strain of Turkey wheat which is practically immune and of very high quality has recently been developed in Oregon. This wheat is well adapted to the dry farming wheat belt of Oregon east of the Cascade Mountains. As this goes to press there is some information available indicating that while the above mentioned varieties of wheat are resistant or immune to the strain of *Tilletia tritici* prevalent in the Pacific Northwest they are apt to prove susceptible to other strains of smut, particularly to *Tilletia laevis*.

**Symptoms.** — Unlike the loose smuts, the bunt does not destroy the glumes. When smutted heads are mature the lemma and palea usually stand somewhat open so that the smutted kernels are slightly exposed to view (Fig. 113). The infested grains or "smut balls" can then be recognized by their color which is darker than that of the sound grains.

Diseased grains retain more or less the shape and size of normal kernels. When broken open the smut balls are found to be filled with a black or

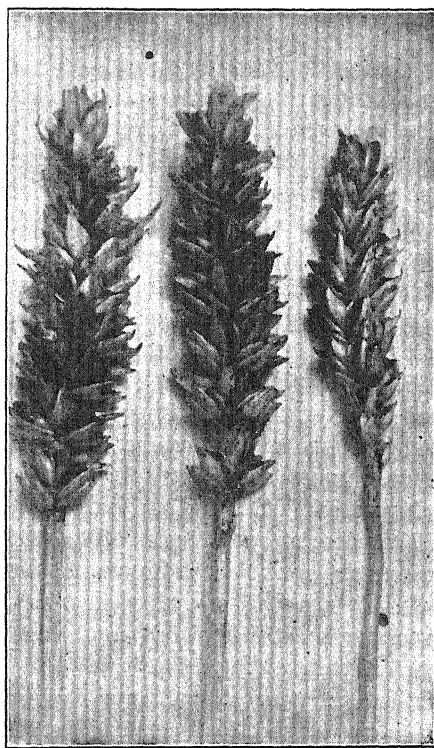


FIG. 113. — Heads of club wheat affected with stinking smut. (After Barss, Ore. Agr. Exp. Sta. Crop Pest Rept. 1915-20. 1921.)

dark-brown powdery substance composed almost entirely of smut spores. A striking characteristic of bunt is the very disagreeable odor, perhaps somewhat like decaying fish. This characteristic has given rise to the common name "stinking smut."

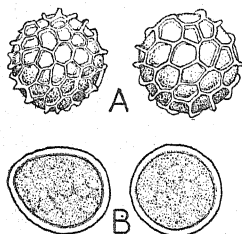


FIG. 114. — Spores of stinking smut. A, *Tilletia tritici*; B, *Tilletia laevis*.

Potter and Coons (23) were able to distinguish between the two species of *Tilletia* which cause bunt, by means of two separate sets of characters. They state that the consistency of the smut mass in *T. laevis* is somewhat oily while that of *T. tritici* is more friable or powdery. They also were able to distinguish the two species on basis of the height of affected plants. The former species does not dwarf the affected plant materially while the latter has a decided dwarfing effect, giving rise to the terms "low smut" for *T. tritici*, and "high smut" for *T. laevis*.

**The causal fungus.** — There are two distinct species of smut fungi causing the disease known as stinking smut. The symptoms caused by these two species have been described above. Their chief morphological difference lies in the spores, there being no marked difference in the vegetative phase of the two species. The vegetative mycelium penetrates the seedling and ramifies throughout the growing part of the plant finally entering the spikelets and the developing ovules. As development proceeds the mycelium forms a dense mass filling the entire inside of the developing grain. Ultimately typical smut spores, chlamydospores, form in the ends of hyphae and in the many short branches which develop in the mass. At maturity, instead of a normal grain there is simply a mass of spores surrounded by a membrane, the mycelium having gelatinized and disappeared to a large extent.

The spores of both species of *Tilletia* average about 16–22  $\mu$  in diameter and are light to dark brown in color. Those of *T. laevis* are smooth on the surface while spores of *T. tritici* are reticulate. The two species can easily be distinguished under the microscope by means of these spore characters (Fig. 114).

On germinating the chlamydospore sends out a non-septate promycelium which produces a cluster of terminal sporidia (Fig. 115). Secondary sporidia are then often formed. Germ tubes from germinating sporidia penetrate the host and bring about infection.

**Perpetuation.** — Chlamydospores, clinging to the seed or left in the soil, carry the fungus over from one crop to the next. Seed-borne smut spores retain their vitality until sowing time on either fall or spring-sown grain. These spores survive in the soil sometimes until winter wheat

is seeded but will not survive the winter in the soil, especially if the smut balls are broken. Spores in unbroken balls have been found to survive until spring in some cases.

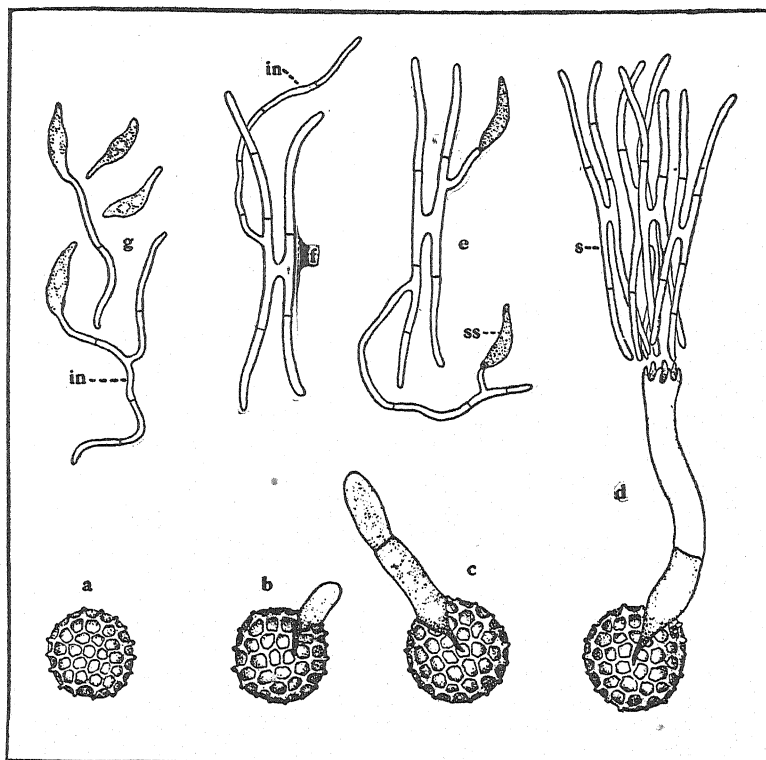


FIG. 115. — Germinating spores of *Tilletia tritici*, one of the species causing stinking smut. (After Heald and Woolman. Wash. Agr. Exp. Sta. Bul. 126.)

**Dissemination.** — Bunt spores are carried on seed wheat or scattered by the wind at harvest and threshing time. In the Pacific Northwest the smut spores are disseminated by the wind at threshing time in such quantities as to give rise to the term "smut showers." Clouds of spores are thus scattered far and wide over the summer fallow fields.

**Infection.** — Infection takes place on the very young seedling, before the first leaf emerges, either from spores carried on the seed or from spores in the soil which may come in contact with the seedling.

**Control.** — In discussing control measures for the stinking smut of wheat two items must be taken into consideration. These are: (a) the fact that the smut spores are borne on the surface of the seed; and (b)



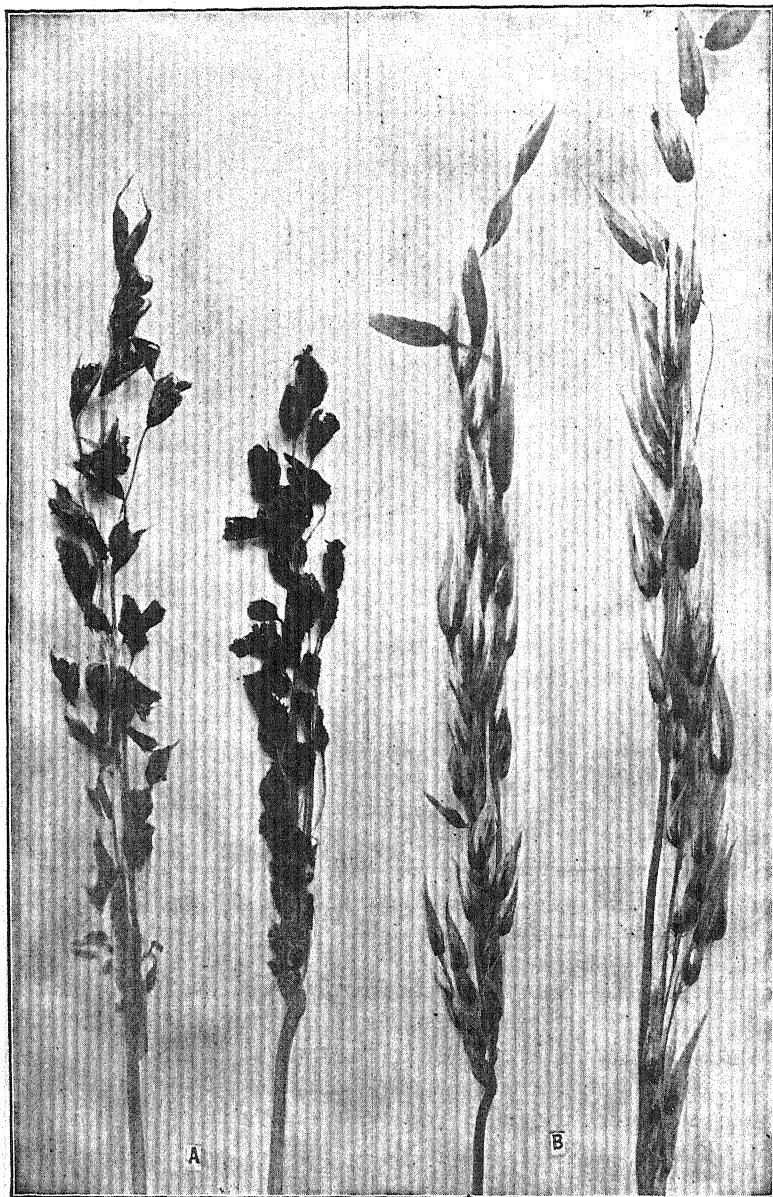


FIG. 116. — Oats smut. A, loose smut caused by *Ustilago avenae*. B, covered smut caused by *Ustilago laevis*. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

soil infestation. The latter item is of no consequence with spring-sown wheat but is an important item with the fall-sown in certain regions.

For the first condition, seed disinfection has long been practiced. Two chief methods of treating seed wheat for bunt are the **copper sulfate** (blue-stone) dip and the **formaldehyde** dip. These have been used for many years. The formula for the copper sulfate bath consists of 1 lb. of copper sulfate dissolved in 5 gals. of water. Seed wheat is placed in wire baskets or bags and immersed in this solution for 3 to 5 minutes. It is then dipped into a bath of lime water, spread out to dry, and planted as soon as possible.

The formaldehyde dip consists of 1 pint of formaldehyde (37 to 40 per cent) to 40 gals. of water. The wheat is soaked in this solution for 5 minutes. After treating, sacked grain should stand for two hours and loose grain should be covered with canvas for a similar time. The grain should then be planted immediately or dried and planted within a few hours.

Both the copper sulfate and the formaldehyde treatments have been objectionable because of the difficulty of avoiding seed injury when these fungicides were used (5, 18, 19). Recently **copper carbonate** dust has been used with great success in controlling stinking smut and with no seed injury. A good grade of the copper carbonate dust should be applied at the rate of two ounces per bushel of seed wheat. Thorough coating of every grain with the dust is necessary for efficient control. A machine of the tumbling churn or revolving drum type

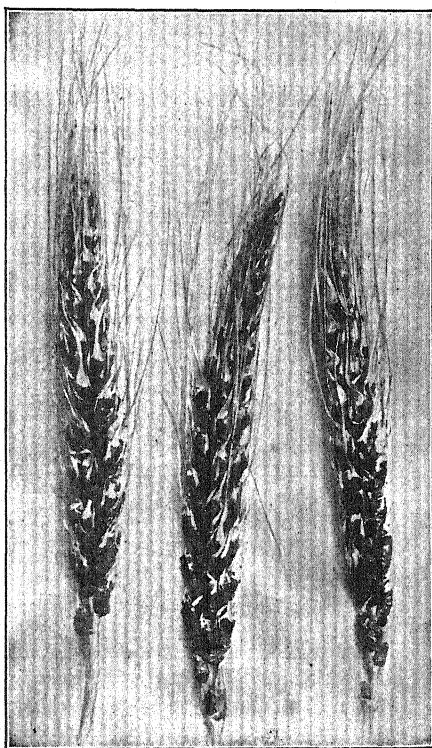


FIG. 117. — Covered smut of barley. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

is very effective in coating every grain thoroughly. Various dusting machines are now on the market. (See reference 16.) Certain mer-

cury compounds such as semisan, germisan and uspulun have given very good results as seed disinfectants for bunt.

The soil infection experienced so extensively in the semi-arid, summer fallow sections of the Pacific Northwest is more difficult to control. Such practices as reploting summer fallow after the smut shower and before seeding, sowing later after the spores have germinated and died, and using smut exhaust fans on separators have been suggested but not generally adopted. The use of spring-sown wheat instead of fall-sown, of course, avoids infection from the soil. But it will not be possible to grow winter wheat entirely free from smut in the Inland Empire of Washington, Idaho and Oregon until resistant varieties are employed, on account of this unavoidable soil contamination.

*Resistant varieties.* — During recent years a great deal of experimental work has been done on the control of smut by means of selecting and breeding for resistance, and very promising results have already rewarded the patience of the scientists who are doing this work. The existence of biologic strains of the bunt fungus complicates the problem, however, and makes its solution more difficult.

*Control of other seedling infection smuts.* — While copper carbonate dust is the best all round seed disinfectant for bunt of wheat, it has not given as uniformly good results when used for covered smut of barley and the oat smuts. For these smuts, the formaldehyde dip gives satisfactory control but causes some seed injury. For hulled oats, Haskell's formaldehyde spray seems to be the best treatment yet devised. This consists in spraying the seed grain with a solution of formaldehyde diluted 1 : 1 with water and applied at the rate of one quart of the diluted solution to 50 bu. of grain, after which the grain is covered with canvas for 4 or 5 hours, then aired, and sown immediately.

#### LABORATORY STUDY OF STINKING SMUT

##### A. Symptoms.

1. *General appearance.* — Compare smutted heads with healthy heads and describe the symptoms exhibited by the whole head. Draw a smutted head and a healthy head side by side to show the difference in appearance. If plants attacked by both species of *Tilletia* are available, compare the two forms. Can you detect any difference? (See reference 23 and this text.)

2. *Smutted grains.* — Remove a diseased grain (smut-ball) and compare with a sound grain. Draw the two side by side. Break open a smut-ball and note the nature of the interior. What is the color and texture of the smut mass? Compare the two species of *Tilletia* in this respect. Can they be distinguished in this way? Draw a smut-ball in longitudinal section.

##### B. Morphology of the fungus.

1. *Mycelium.* — If sections of wheat plant showing mycelium, or illustrations of same, are available, examine and draw.

2. *Spores*. — Mount some of the smut dust in water and examine under the microscope. Of what does this powder consist? Study the spores with high power. Note surface markings and content of spores. Compare the spores of the two species as to size, markings and content. Are they distinguishable? Draw. Find out, if possible, how and where these spores are produced by the smut fungus.

Study germinating spores and note the promycelium and sporidia (basidiospores). Draw.

### C. Life Cycle.

1. *Perpetuation and dissemination*. — Examine sound grains of wheat secured from a batch threshed from a field which had a considerable percentage of smutted plants. Do the grains look perfectly clean? Which end of the grain looks darker in color? Examine this end with a hand lens. Then scrape this part of the grain with a scalpel and examine the scrapings under the microscope. What do you find? What do you conclude with reference to the manner of carrying the smut disease over from one crop to the next?

2. *Infection*. — What is the source of infection in the new crop? When, where and under what conditions does infection by the stinking smut fungus occur? Does infection ever occur even if no spores are clinging to the seed grain when sown? (See text.)

D. *Experimental*. — If time and conditions permit, try this experiment. Get some seed wheat that is badly contaminated with smut spores. Divide this smutty wheat into several lots. Sow one lot without any treatment. Treat another lot with the copper carbonate dust. If desired other lots may be treated with copper sulfate and with formaldehyde. If possible, get some wheat from a source where you know it has never been contaminated at all. Sow all these lots under similar conditions and watch results.

E. *Notes*. — Write a complete account of this disease using information secured from all available sources. Follow the outline on page 152, modifying it if necessary to make it fit this disease.

### REVIEW QUESTIONS

1. To which infection group of smuts does bunt belong? (See page 360 of this text and also Reference 22.)
2. What is the life cycle of the group to which the bunt fungus belongs? State in detail.
3. How does the life history of this smut fungus affect the control measures to be recommended for its control?
4. Under what particular set of conditions is even the most careful and approved seed treatment ineffective for bunt control? (See text and Reference 14.)
5. What cultural practices have been suggested as a means of overcoming the danger from soil infestation? Are these practical enough to permit of general adoption among wheat growers?
6. Is soil infestation a problem in spring-wheat sections? Why?
7. What difficulties have been experienced in the past with the commonly used liquid seed treatments? (See References 5, 18 and 19.)
8. If a grower knows that his soil is infested, is it worth while for him to treat his seed before sowing? Why?
9. What headway has been made along the line of securing varieties of wheat resistant to bunt? (See Reference 27.)

10. What practical difficulties must be overcome in developing smut-resistant wheats? (See Reference 27.)
11. What advantages has the copper carbonate dust over the formaldehyde and blue-stone treatments?
12. Discuss the relation of soil moisture and soil temperature to bunt infection in wheat. (See Reference 17.)

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### Loose Smut of Wheat

Caused by *Ustilago tritici* Eriks.

This smut is a typical example of the blossom infection group. In fact, since the work of Tisdale and others has demonstrated that the loose smut (*U. nuda*) of barley is, in part at least, a seedling-infection smut, the loose smut of wheat is the only one of the cereal smuts listed above which is strictly of the blossom infection type so far as known at present. Loose smut of wheat is easily distinguished from stinking smut and flag smut of the same grain, being characterized by the complete destruction of spikelets, whereas bunt is confined to the kernels, leaving the glumes intact, and flag smut attacks the leaves and culms. Loose smut is sometimes called "black heads," "blasted heads" or "snuffy ears."

**History and distribution.** — Prior to 1888 no clear distinction had been made between the loose smuts of wheat, barley and oats. Various names had been used at different times to designate them but the same name was always applied to all the loose smuts. Jensen (6, 7) showed that the two barley smuts, covered and loose, were distinct and also

that the loose smut of wheat could not infect any other species of cereal. Maddox (9, 10) first proved the true nature of the loose smut of wheat in 1895 when he demonstrated blossom infection in that smut. He included loose smut of barley in the same classification and since that time this smut has been considered as belonging in the blossom infection group until recently when evidence was brought forth to indicate that the latter smut is also a seedling infection smut, under certain conditions at least (see reference 7 on page 383). Maddox's work was confirmed in 1903 by Brefeld who, apparently without any knowledge of the work previously done by Maddox, independently proved that blossom infection occurs in both the loose smut of wheat and the loose smut of barley. When blossom infection was first discovered it was supposed that spores were carried over, imbedded in the seed, and caused the next year's crop to be smutted, but later it was found that the smut fungus hibernates in the form of mycelium instead of spores.

The loose smut of wheat occurs in practically all wheat-growing regions of the world. It is well known in Australia where Maddox did his work on blossom infection. It occurs in varying amounts in many provinces of India and is well known in Europe. In the United States it is encountered wherever wheat is grown but is found in serious amount for the most part in certain sections east of the Rocky Mountains. In many parts of the country, especially the far West, only a trace is occasionally found.

**Economic importance.** — In many regions the loose smut of wheat is of little economic importance because it occurs in such small amounts. However, there are sections where the damage is considerable. As a rule, the losses from loose smut in the United States are not as great as those resulting from stinking smut but there are seasons and localities in which the loose smut of wheat causes more damage than bunt. The Plant Disease Reporter (Supplement 30) records an estimated reduction of 1.1 per cent in yield due to loose smut in the United States in 1922, or a loss of 10,484,000 bu. States suffering the greatest losses that year were Illinois, 3 per cent or 2,040,000 bu.; Ohio, 2 per cent or 1,004,000 bu.; and Indiana, 3 per cent or 1,036,000 bu. In 1923 the total loss from this disease in the United States according to the Plant Disease Reporter (Supplement 36) was 9,964,000 bu., while in 1924 the same authority (Supplement 43) reported a loss of 9,800,000 bu. for the entire country. In 1924 the loss in North Dakota was 2 per cent or 1,524,000 bu., and in Kansas, 1.2 per cent or 1,095,000 bu. These figures indicate that in many of the wheat-growing states east of the Rocky Mountains, loose smut is a serious disease and in some cases ranks with bunt in the amount of losses caused.



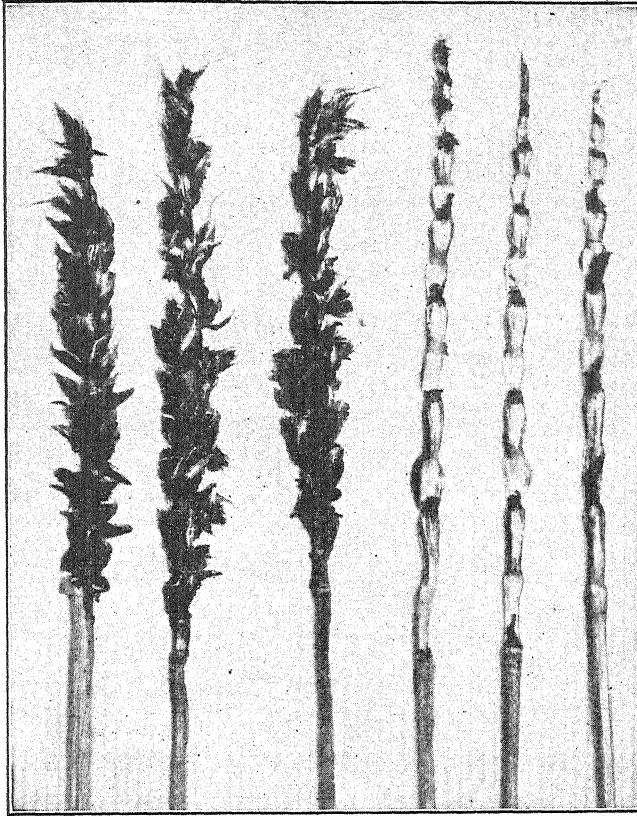


FIG. 118. — Loose smut of wheat. (After Jackson, Ore. Agr. Exp. Sta. Crop Rept. 1911-12.)

**Symptoms.** — The most striking symptom, and the only one usually noticed, consists of the black smutty appearance of the head as it emerges from the sheath. The glumes and kernels are completely disintegrated and soon after emergence the wind blows this powdery mass away leaving only the bare rachis which is not attacked by the fungus (Fig. 118). Usually all spikelets in a head are destroyed but occasionally only a part of a head is affected. The black powdery mass consists largely of spores. The smutted heads appear shortly before the heads emerge in the normal plant and the spores are mature and are disseminated at the time the healthy heads are in bloom. Abnormal cases have been reported (6) in which this smut occurred on the leaf or culm. The symptoms of loose or naked smut of barley (Fig. 120) and loose smut of oats (Fig. 116 A) are very similar to those of the loose smut of wheat.



The smut masses in all three of the loose smuts are at first inclosed in a delicate grayish membrane which breaks either before or after the head emerges from the sheath.

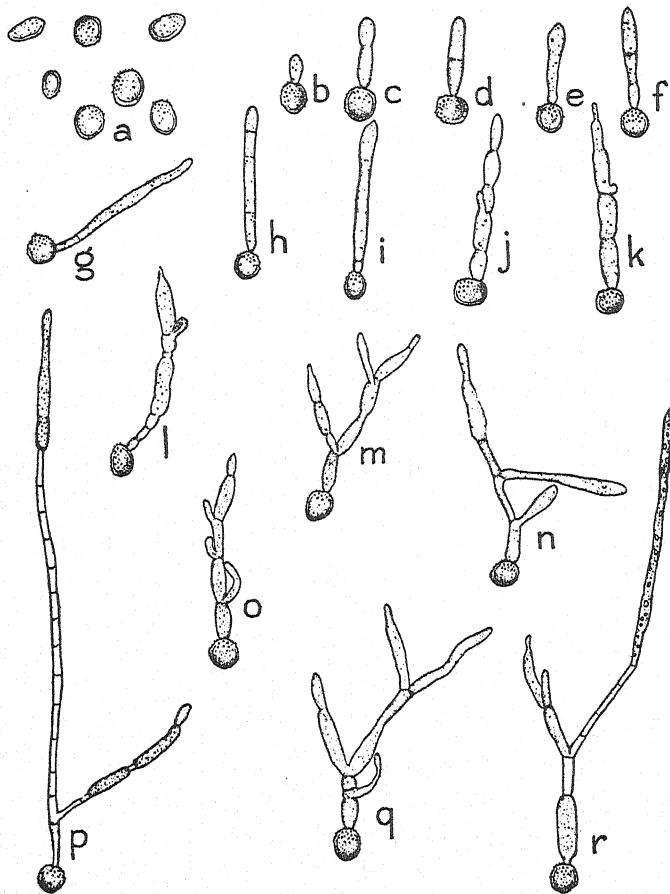


FIG. 119. — Germinating spores of *Ustilago tritici*. (After Stakman.)

**Morphology and life history of the fungus.** — The mycelium of the loose-smut fungus develops within the embryo of the maturing seed without causing any outward symptoms. The ripened seed seems normal in every respect. The fungus hibernates in the kernel in the form of dormant mycelium. When the seed germinates the mycelium again becomes active, keeping pace with the growth of the young plant, and is carried up in the growing point so that when the head begins to form it is soon invaded by the mycelium. The infested heads are soon

destroyed by the action of the fungus and then innumerable spores are formed from the massed mycelium. Most of the black powdery mass thus produced consists of spores called chlamydospores. These spores are more or less spherical in shape, minutely echinulate, and measure 5 to 9  $\mu$  in diameter (Fig. 119 a). The spores in the diseased heads are mature at the time the healthy heads are in blossom and are wind-disseminated. Some spores lodge between the glumes of healthy heads and come in contact with the feathery styles. Spores of *Ustilago tritici* germinate by sending out promycelia, a general characteristic of smuts, but few if any sporidia are produced (Fig. 119). The promycelia act as germ tubes and penetrate the styles. They then grow down into the ovary and enter the ovules where a mycelium develops in the embryo of the young seed thus completing the life cycle.

**Control.** — In devising control measures for the loose smut it is necessary to remember the salient facts of the life history of the causal fungus. Since the fungus hibernates as dormant mycelium imbedded within the seed it is evident that fungicides in the form of dips or dusts such as are used for bunt and the other seedling infection smuts can not be effective in controlling this smut. The only effective seed treatment thus far developed for loose smut of wheat is the hot water bath. This consists essentially in dipping the seed wheat for a sufficient time in water hot enough to kill the imbedded mycelium but not hot enough to kill the seed. Jensen first worked out a successful method of applying this treatment. This method, slightly modified, is as follows:

*Hot-water seed treatment for loose smut.* — (a) Soak the seed grain in cold water for 4 to 6 hours. (b) Place in warm water kept at about 120° F. for 1 minute. (c) Immerse in water held at 129° F. for 10 minutes. (d) Remove and dip in cold water to stop the action of the heat. (e) Spread out to dry. Plant as soon as dry enough, or dry out thoroughly and store until seeding time. Barley may be treated for loose smut by the same method except that it should be dipped in water held at 126° F. for 13 minutes. The hot water method requires great

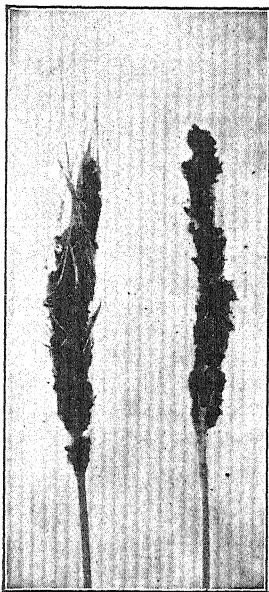


FIG. 120. — Loose smut of barley. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept. 1911-12.)

care and is not popular with farmers in general. In some places a community plant is maintained where all the farmers in the neighborhood may bring their grain to be treated. In regions where loose smut is very prevalent it becomes a factor in the profitable production of wheat and it is advisable to treat at least enough seed for sowing the special seed plot if not for the general commercial plantings.

*Varietal resistance.* — While some varieties of wheat are less susceptible to loose smut than others no outstanding examples of resistance to this smut have been found. Work along this line has not proceeded as far as in the case of bunt nor with such marked success in selecting and breeding resistant strains.

#### LABORATORY STUDY OF LOOSE SMUT OF WHEAT

1. *Symptoms.* — Compare the symptoms of this smut with those of bunt previously studied. How do they differ both as to effect on the glumes and on the grains? Sketch to show characteristic symptoms.

2. *Morphology and life cycle of the fungus.* — Mount spores and compare with bunt spores as to size and markings. *Draw.* Examine germinating spores and compare with germinating spores of *Tilletia* (Fig. 115). Also compare with germinating spores of the corn smut fungus, *Ustilago zeae* (Fig. 123), and the oat smut fungus, *Ustilago avenae*. *Draw* germinating spores.

*Life cycle.* — Learn the difference between the life histories of the seedling-infection smuts and the blossom-infection smuts. To which group does the loose smut of wheat belong? What difference does this make in the methods of controlling the two smuts? Compare the loose smut of barley with this smut. (See reference 2.)

3. *Notes.* — Write a complete description of the symptoms, life cycle, and methods of control of the loose smut of wheat, comparing it in all respects with the stinking smut of wheat.

#### REVIEW QUESTIONS ON LOOSE SMUT OF WHEAT

1. Describe the symptoms of loose smut of wheat. Compare with bunt as to effects on various parts of the plant.

2. To which infection group does this smut belong? Give the life history of this group. What other smut belongs in this group?

3. Why is not seed disinfection with copper carbonate, formaldehyde or copper sulfate effective in controlling this smut?

4. What principle is involved in the hot-water treatment?

5. Describe the hot-water treatment in detail and explain the precautions that must be taken to make it a success.

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### Corn Smut

Caused by *Ustilago zeae* (Beck.) Ung.

**Historical.** — The earliest record of corn (*Zea mays*) smut (2) was published by Bonnet, a Frenchman, in 1754. Corn is a native of America and was introduced into Europe probably as early as 1500. No one seems to know whether or not the corn smut originated in the native home of the corn plant but presumably it did. The earliest record of corn smut in America was made in 1822 by Schweinitz. Roulin reported in 1829 that he had observed this disease in South America.

Kühn observed the germination of the spores of this smut in 1857, and in 1874 he saw the germ tubes penetrate the epidermis of the corn plant. It remained for Brefeld (3) to work out the true life history of the corn smut fungus. This he published in 1895 along with the results of his researches on some of the other grain smuts.

**Geographical distribution.** — Corn smut appears to occur to a greater or less extent wherever corn is grown. In the United States it is most prevalent, perhaps, in the great corn-growing belt of the central states both east and west of the Mississippi river. However, there is scarcely a state in the Union in which at least a trace of corn smut does not occur nearly every year, with now and then a bad year in which considerable loss occurs in some sections of the country.

**Economic importance.** — The "Plant Disease Reporter" (1) gives a summary of losses due to corn smut in the various states for the years

1918 to 1924 inclusive. A few examples taken at random will serve to show the losses incurred in these years. During the year 1918 the loss in Connecticut was 0.1 per cent of the crop; New York, 1 per cent; West Virginia, 4 per cent; Georgia, 5 per cent; Texas, 8 per cent; Illinois, 1.5 per cent; Iowa, 2.5 per cent; South Dakota, 5 per cent;

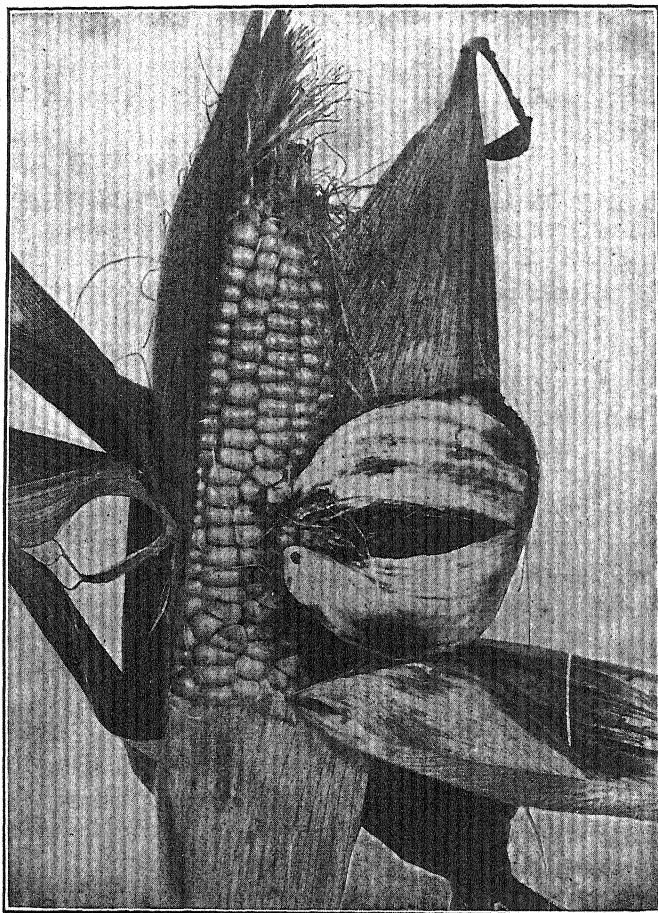


FIG. 121. — Common smut of corn. Symptoms resulting from ear infection. (After Jackson, Del. Agr. Exp. Sta. Bul. 83.)

Nebraska, 5 per cent; Kansas, 8 per cent; Colorado, 30 per cent; Oregon, a trace; California, a trace. The total loss in the United States for 1918 was estimated at 70,876,000 bu. In 1921 the same states lost as follows: Connecticut, 1.5 per cent; New York, 1 per cent; West Virginia, 2 per cent; Georgia, 2 per cent; Texas, 3 per cent; Illinois,

3.5 per cent; Iowa, 1.5 per cent; South Dakota, 15 per cent; Nebraska, no figures; Kansas, 5 per cent; Colorado, 2.5 per cent; Oregon, trace; California, 10 per cent. For 1921 the total loss in the United States was 104,533,000 bu.

**Hosts.** — The smut under discussion, the common or boil smut of corn (*Ustilago zaeae*), attacks only the corn plant, *Zea mays*. It should

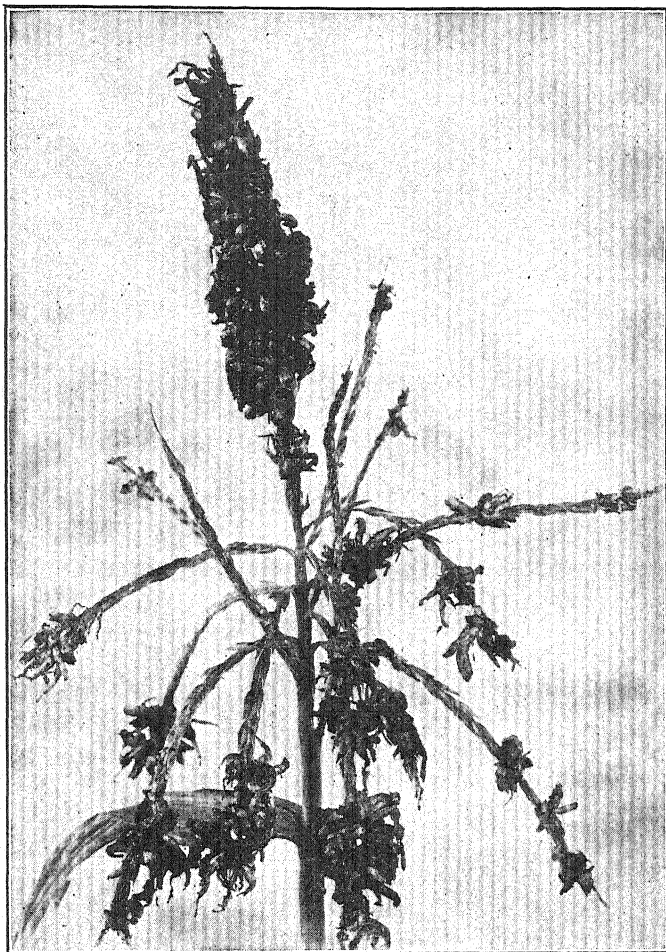


FIG. 122. — Common smut of corn in the tassel. (After Jackson, Del. Agr. Exp. Sta. Bul. 83.)

be mentioned that another species of smut fungus, *Sorosporium reilianum*, attacks corn occasionally causing a "head smut" which, by the uninitiated, might be confused with the common boil smut but which

is by no means as prevalent as the common smut and is encountered only occasionally in certain localities. The head smut attacks sorghum as well as corn and is considered a more serious disease of the former than of maize.



FIG. 123. — Germinating spores of *Ustilago zeae*. (After Stakman.)

**Symptoms.** — Any part of the plant above ground may be attacked, provided the part is in a tender growing condition. The symptoms may appear, therefore, on ear (Fig. 121), tassel (Fig. 122), stem or leaf. The infection is local and a single kernel or floret may show the symptoms, or every kernel on the ear and every floret on the tassel may be affected.

Large, boil-like abnormalities or swellings occur which may attain to several inches in diameter, especially on the ear. At first these tumors are covered with a white membrane and the central part consists of fungous mycelium. At maturity the central portion appears as a black or dark-brown powdery mass consisting largely of smut spores. The covering membrane becomes dry, breaks and the spores are disseminated. Similar tumors appear on the leaves and on the stem, especially at the nodes.

**Life Cycle.** — The irregular, branching mycelium grows through or between the cells in local areas. When the tumors produced are mature the mycelium forms large numbers of short irregular branches which swell, and portions of them round off as spores. When spore production is complete the mycelium has largely disintegrated so that the smut boil is filled with the chlamydospores and fragments of broken-down mycelium.

The chlamydospores are capable of immediate germination but may remain viable for one or more years before germinating. Thus the disease may be perpetuated on old smutted plants left in the field, on fodder, in manure, etc. On germination a chlamydospore produces a promycelium which in turn buds off sporidia or basidiospores (Fig. 123). These basidiospores are wind-disseminated, and if they lodge on the growing parts of a corn plant under proper conditions infection may take place.

**Control.** — It has been proved that the corn smut is not seed-borne and that infection is local rather than systemic. Therefore, seed disinfection is of no avail. Since the fungus is perpetuated on old stalks in the field, on fodder and in manure it follows that *sanitation* and *crop rotation* are the best control measures available until resistant varieties are developed. Smut resistance has been observed in some strains of corn but the many biologic forms of *Ustilago zeae* (10) complicate the problem of control by means of resistant varieties. However, some headway is being made on this problem.

#### LABORATORY STUDY OF CORN SMUT

**A. Symptoms.** — Examine specimens of smutted corn and note symptoms. On what parts of the plant do the symptoms occur? Are the effects of the disease similar on all parts attacked? Make **drawings** to illustrate all symptoms observed.

**B. Morphology and life cycle of the fungus.** — Mount some of the black powder from a smut boil and examine with the microscope. Of what does the smut dust consist? Compare the spores of the corn smut fungus with the spores of other species of smut fungi studied. **Draw.** Germinate spores of *Ustilago zeae*. Note the production of promycelium and sporidia. Compare this manner of germination with that of the bunt fungus and of any other smut fungi studied. **Draw.** Look up the method of perpetuation, dissemination and infection.



C. Notes. — Write a complete account of the common smut of corn, including especially the symptoms, life history and control.

#### REVIEW QUESTIONS

1. To which infection group does the corn-smut fungus belong? Give the life cycle.
2. How does the type of infection occurring in this smut affect the recommendations for control?
3. Why is not seed treatment effective for control of corn smut?
4. What fact complicates the problem of control by means of resistant varieties?
5. Distinguish between the common smut and the head smut of corn.

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### The Rusts

The rusts (Uredinales) constitute a very large and extremely destructive order of the basidiomycetes. The group consists of some two thousand species, many of which are of great economic importance. A few of the well-known species are: black stem rust of grains and other grasses (*Puccinia graminis*), leaf rust of wheat (*Puccinia triticea*), crown rust of oats (*Puccinia coronata*), clover rust (*Uromyces trifolii* and *U. fallens*), bean rust (*Uromyces appendiculatus*), cedar rust (*Gymnosporangium juniperi-virginianae*), white pine blister rust (*Cronartium ribicola*), carnation rust (*Uromyces caryophyllinus*) and hollyhock rust (*Puccinia malvacearum*).

**Spore forms.** — Some species of rusts exhibit a larger number of spore forms and a more complicated life history than are found in any other group of fungi. A rust species which exhibits all known spore forms, produces at various stages in its life cycle a total of five different types of spores as follows: (a) pycniospores (spermatia), usually borne in a somewhat flask-shaped structure known as a pycnium (spermogonium); (b) aeciospores (aecidiospores) usually borne in a more or less cup-like structure known as an aecium (aecidium); (c) urediniospores (uredospores), borne in a pustule or sorus known as a uredinium (uredo-sorus); (d) teliospores (teleutospores), borne in a telium (teleutosorus); and (e) basidiospores (sporidia) borne on a basidium (promycelium) arising from the germinating teliospore.

Some species of rusts do not exhibit all of these spore forms in their life history. The missing spore form varies in different species. Sometimes the uredinial stage is not present. In other cases pycniospores or aeciospores are lacking. Certain species have only one spore form present, the teliospore. In taxonomic works on the rusts the following symbols are generally used to designate the various spore-stages occurring in the life cycle of the rust:

O = Pycnia; I = Aecia; II = Uredinia; III = Telia. Since basidiospores always arise from the teliospore it is not necessary to consider them in a scheme of this kind.

In view of the fact that various species of rusts exhibit various combinations of spore forms present or missing it will be of interest to indicate all the different combinations found in the group. The following types were suggested by Schröter.

Eu-type: O, I, II, III present.

Brachy-type: O, II, III present; I omitted.

Opsis-type: O, I, III present; II omitted.

Hemi-type: II, III present; O, I omitted.

Micro-type: only III present; germination only after a resting period.

Lepto-type: only III present; germination immediate.

**Heteroecism.** — A peculiar and interesting thing about the life history of many of the rusts is the fact that not all of the spore forms occurring in the cycle are produced on the same host. There are many cases in which some of the spore forms occur on one host and the remaining forms are found on one or more quite distinct species of plant. For example, in the black stem rust of wheat the urediniospores and teliospores occur on the wheat plant while the pycniospores and aeciospores are formed on the common barberry bush. The basidiospores arise from the germinating teliospore and therefore are always closely associated with the telial host. When a rust thus completes its life cycle on two alternating hosts it is said to be *heteroecious*. The barberry bush, on which the pycnial and aecial stages of the heteroecious black stem rust of wheat occur, is known as the *alternate* or *complementary* host. However, if all of the spore forms are produced on the same species of host plant and the rust thus does not alternate between two different species of host plants in completing its life cycle, it is said to be *autoecious*.

Referring again to the different types previously mentioned it is evident that the first three types, the eu-type, the brachy-type and the opsis-type may be either autoecious or heteroecious. The hemi-, micro-, and lepto-types are always autoecious. Where only urediniospores and teliospores occur in the life cycle of a rust they are always

associated together on the same host. It is self-evident that where only one spore form occurs, as in the micro- and leptotypes, there can be no alternate host.

**Function of the different spore forms.** — Each form of spore performs a definite and special function. Especially is this true of the heteroecious species.

The function of the *pycniospore* has always been a matter of doubt. In the past, two possible explanations of the function of these minute spore-like structures have been advanced. One is that they are degenerate male gametes (hence the term spermatia formerly applied to them), and the other is that they are conidia. In either case the opinion has generally prevailed in recent years that the pycniospores are probably functionless. However Craigie (29,\* 30\*) has recently produced evidence to show that these spore forms probably do have a function. He states that in certain rusts, *Puccinia helianthi* and *Puccinia graminis*, he finds evidence of heterothallism and that some of the basidiospores are plus and some minus in nature. Plus basidiospores give rise to mycelium which produces plus pycniospores, and minus basidiospores give rise to mycelium which produces minus pycniospores. It is only when pycniospores from plus and minus pustules are intermingled that aeciospores are produced. If no inter-mixing of spores from plus and minus pycnia occurs, only pycniospores are produced.

The *aeciospore* (Fig. 127), is a functioning spore, which, in case of the heteroecious species, always infects the telial host and seldom, if ever, can re-infect the host on which it was produced. In autoecious species, however, the aeciospore re-infects the host type which produced it.

*Urediniospores* (Fig. 128) are repeating spores which always infect the same host type as that on which they grew, never an alternate host. They serve as a rapid means of spread during the summer between individuals of the same host type.

*Teliospores* (Fig. 129) are frequently overwintering forms and in germinating always give rise to *basidiospores*. In case of heteroecious forms the basidiospores always cause infection on the alternate host, never on the host which produced the teliospore. In autoecious forms, of course, the basidiospore re-infects the telial host.

Thus in a heteroecious eu-type rust there are two spore forms which can cause infection on the telial host, namely, aeciospores and urediniospores, while only one spore form can infect the aecial host, namely, the basidiospore. In an autoecious eu-type rust three different kinds of spores can cause infection on the one host, namely, aeciospores, urediniospores and basidiospores.

\* See foot of page 401 and top of page 402.

**Sexuality in the rusts.** — For a long time the sexual process in rusts as well as in other basidiomycetes was obscure and doubtful. Since 1904, however, it has been discovered that a binucleate condition arises in the base of the aecium and persists throughout the aeciospores, the urediniospores and the young teliospores. In the maturing teliospore the two nuclei fuse, again bringing about a uninucleate condition. This whole process may be considered a sex act. For a more complete discussion of the cytology and sexuality of the rusts see the following references. (3, pp. 17-29, and 4, pp. 196-218.)

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## The Cereal Rusts

Probably the rust diseases which have been known for the longest time and the ones most often mentioned in the early history of plant diseases are those which attack the grains, wheat, oats, barley and rye. The following outline includes all known rusts found on these cereals and indicates the alternate host where known.

## Cereal host and name of rust.

## Alternate host.

## WHEAT

Stem rust caused by

*Puccinia graminis tritici* . . . . . Common barberry, *Berberis vulgaris*.

Leaf rust caused by

*Puccinia triticea* . . . . . Meadow rue, *Thalictrum* spp.

Stripe rust caused by

*Puccinia glumarum* . . . . . Unknown.

## BARLEY

Stem rust caused by

*Puccinia graminis tritici* . . . . . Common barberry, *Berberis vulgaris*.

Stem rust caused by

*Puccinia graminis secalis* . . . . . Common barberry, *Berberis vulgaris*.

Leaf rust caused by

*Puccinia simplex* . . . . . Star of Bethlehem, *Ornithogalum umbellatum*.

Stripe rust caused by

*Puccinia glumarum* . . . . . Unknown.

## RYE

Stem rust caused by

*Puccinia graminis secalis* . . . . . Common barberry, *Berberis vulgaris*.

Leaf rust caused by

*Puccinia dispersa*.....Bugloss, *Anchusa (Lycopsis) arvensis*.

Stripe rust caused by

*Puccinia glumarum*.....Unknown.

#### OATS

Stem rust caused by

*Puccinia graminis avenae*.....Common barberry, *Berberis vulgaris*.

Leaf or crown rust caused by

*Puccinia coronata*.....Buckthorn, *Rhamnus* spp.

It will be noticed that strains or varieties of the stem-rust fungus attack all four of these grains, there being a distinct strain for each of the grains, wheat, oats and rye, while barley has no distinctive strain of its own but is susceptible to both the wheat strain and the rye strain of the stem rust. On the other hand, there is a distinct species of leaf-rust fungus on each of these four cereals. The stripe-rust fungus attacks all of these grains except oats. It has been demonstrated that some of the above species or strains of rust fungi are composed of many sub-strains or biologic forms. This fact will be discussed further in the following discussion of stem-rust. Only one of the species of cereal rusts can be taken up in detail here but a number of references on the other species will be found immediately following the list of references on stem-rust.

### Black Stem-rust

Caused by *Puccinia graminis* Pers.

**Historical.** — Rusts of various crops are mentioned in ancient history, and while it is probable that all the diseases known as rusts in early times were not true rusts, yet it is certain that the true rusts of the cereal crops did occur and cause great losses in ancient times as well as in modern times. The writings of the Romans bear evidence that their farmers were familiar with the rust attacks on their grain crops and feared this disease. Roman mythology bears evidence of this in the fact that these people evolved a special rust-god pair, Rubigus and Rubigo, to whom they prayed for relief from this pest. While it is likely that more than one species of rust occurred on the various cereal crops in ancient times just as we recognize several species today, it is probable that the stem-rust was one of the most important then as it is now.

In more modern times we find evidence of the prevalence of rust in grain fields. At Rouen, France, in 1660, a decree was promulgated against the growing of the barberry bush because observation and

experience led the people to believe that this plant bore some mysterious relation to outbreaks of wheat rust. Again we learn that, about the year 1805, grain rust was serious and attracting a great deal of attention in Denmark. Here also the peasants noticed that the rust was more severe in the vicinity of barberry bushes. Differences of opinion as to the responsibility of the barberry led to conflicts between those who would destroy this plant and those who wished to retain it in their gardens and hedges.

The true life history of the black stem-rust fungus was not known until 1865 when De Bary, by inoculation work, succeeded in demonstrating beyond a doubt that one stage in the life history of this rust fungus does occur on the barberry and thus established the facts underlying the phenomena which up to this time had been surrounded by mystery and superstition. Since the classic work of De Bary opened the way this species of rust fungus has been the subject of almost innumerable researches and investigations dealing with various phases of its morphology and life history, the existence of biologic forms or races, the question of varietal resistance and susceptibility, etc.

**Geographical distribution.** — The black stem-rust occurs in practically all countries of the world where grain is grown, including the United States, Canada, South America, Australia, Europe, Africa and Asia. In the United States it is most serious in the great wheat belt of the northern Mississippi Valley and Plains States. It is also severe in the Gulf States.

**Economic importance.** — The historical accounts previously mentioned indicate that for thousands of years the damage caused by rust has attracted much attention among agriculturists wherever the cereals were grown. Evidently great losses were occasioned by rust during certain years in ancient times as well as at the present time. In the United States the year 1904 was marked by a severe epidemic of stem-rust. In that year, the loss in the Dakotas and Minnesota is estimated at \$20,000,000. Again, in 1916, another outbreak occurred when the yield of wheat was reduced at least 180,000,000 bu. in the United States and 100,000,000 bu. in Canada. The report issued by The Plant Disease Survey (26), covering the year 1922, estimates the losses from stem-rust in North Dakota at over 14,000,000 bu.; in South Dakota, somewhat over 1,000,000 bu.; and in Minnesota about 1,500,000 bu. The same authority (27) estimates the losses in 1923 as follows: Minnesota, 15 per cent or 3,118,000 bu.; North Dakota, 12 per cent or 7,039,000 bu.; South Dakota, 10 per cent or 2,691,000 bu.; Montana, 18 per cent or 9,447,000 bu.

**Hosts.** — This species is known to occur on nearly one hundred species of the grass family, including wheat, oats, barley, rye, spelt, emmer, timothy and other forage grasses, as well as a large number of wild species of grasses, such as the wheat-grasses, rye-grasses, wild-barleys, fescue-grasses and brome-grasses. The aecial stage occurs chiefly on the common barberry, *Berberis vulgaris*, but may occasionally be found on certain other species of barberry or Mahonia.

*Susceptibility of varieties and biologic races.* — While the species, *Puccinia graminis*, as a whole, can attack any of the nearly one hundred species of the grass family mentioned above, yet it is true that, as a rule, much fewer than that number of species are found to be susceptible in any particular locality. This has been found to be due to the fact that the species of rust fungus causing black stem-rust is composed of many different biologic forms, races or varieties. Any particular form or variety of the rust fungus can attack certain species or varieties of host plants but not all of the hundred species. Thus the following specialized varieties of *Puccinia graminis* may be cited as examples of this condition.

Biologic form	Hosts
<i>Puccinia graminis tritici</i> . . . . .	Wheat, barley and certain wild grasses.
<i>Puccinia graminis secalis</i> . . . . .	Rye, barley and certain wild grasses.
<i>Puccinia graminis avenae</i> . . . . .	Oats and certain grasses.
<i>Puccinia graminis phlei-pratensis</i> . . . . .	Timothy and certain other grasses.
<i>Puccinia graminis agrostis</i> . . . . .	Redtop and other grasses.
<i>Puccinia graminis poae</i> . . . . .	Bluegrass and other grasses.

Again further research has shown that the race on wheat, *P. graminis tritici*, is divided into sub-races or forms some of which can attack certain varieties of wheat, others other varieties, but no one of which can attack all varieties of wheat with equal facility. Stated conversely, this means that certain varieties of wheat are susceptible to one sub-race or form of the wheat strain of the rust while certain other varieties of wheat are more susceptible to some other form of the rust fungus. Furthermore, it has been discovered that different races of the rust are not all found in the same region so that a certain variety of wheat which may be resistant to the races of rust found in one section of the country, for example, Minnesota, may be susceptible to the forms found in another region, as, for example, Texas. It can readily be seen that this fact very seriously complicates the problem of finding varieties of wheat that are resistant to stem-rust since a variety which is satisfactorily resistant in one part of the country may be very susceptible in another region. Stakman and his co-workers have differentiated a large number



of strains of *Puccinia graminis tritici*. In 1917 Stakman and Piemeisel (15) reported the first sub-race of the wheat stem-rust and named it *Puccinia graminis tritici compacti*. In 1919 Stakman, Levine and Leach (20) reported that up to October 1, 1918, a dozen forms had been discovered by inoculation experiments carried on at the University of Minnesota. In 1922 Stakman and Levine (21) stated that no less than thirty-seven biologic strains of *P. graminis tritici* had been differentiated up to that time.

**Symptoms and signs.** — On the grain or grass hosts there are two different stages which we should be able to recognize. In the early summer the first stage appears. This is variously known as the summer stage, red rust stage or uredinal stage. It may be recognized as elon-

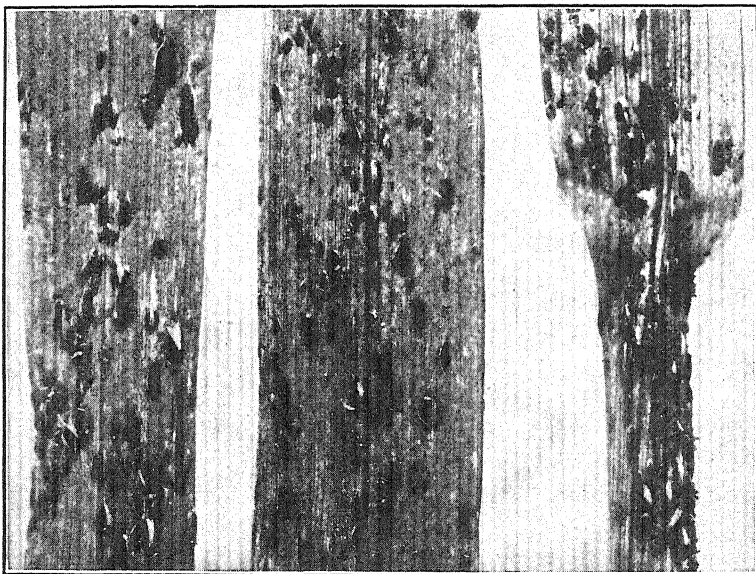


FIG. 124. — Uredinia of stem-rust on wheat.  $\times 4$ . (After Gilbert, S. D. Agr. Coll. Ext. Cir. 33.)

gated pustules or blisters of varying size, sometimes reaching several millimeters in length (Fig. 124). At first the epidermis is raised in long, narrow elliptical shaped blisters parallel with the long axis of the stem or leaf, mostly on the leaf sheath which surrounds the stem. As these pustules (called uredinia) reach maturity the epidermal covering splits open in an irregular manner so that there is an elongated crater-like opening with irregular flaps of the epidermis clinging to one or both sides of the opening. Within these open pustules are masses of reddish

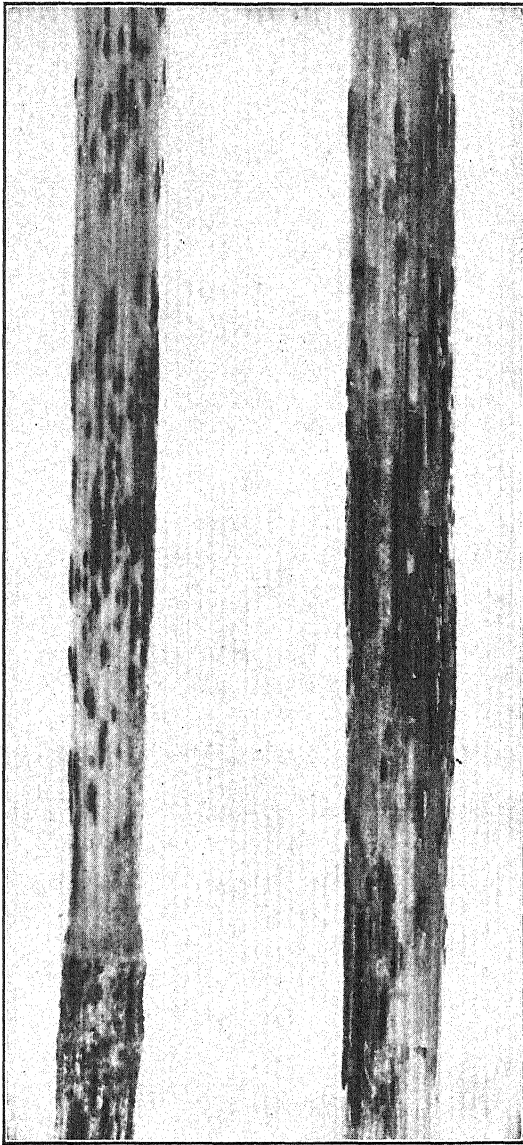


FIG. 125. — Telia of stem-rust on wheat.

or rust-colored powder consisting of thousands of minute dust-like spores (see under Life History). Later in the season the black, or winter stage appears. This consists of elongated pustules (telia) similar in shape to the red pustules but black in color (Fig. 125). The black color

is due to the fact that the spores in these pustules are darker in color than those of the red stage so that when a large number of them are massed together the color of the mass is black. It should be kept in mind that the most conspicuous character which distinguishes this rust from all other species of rust occurring on grains and other grasses is the

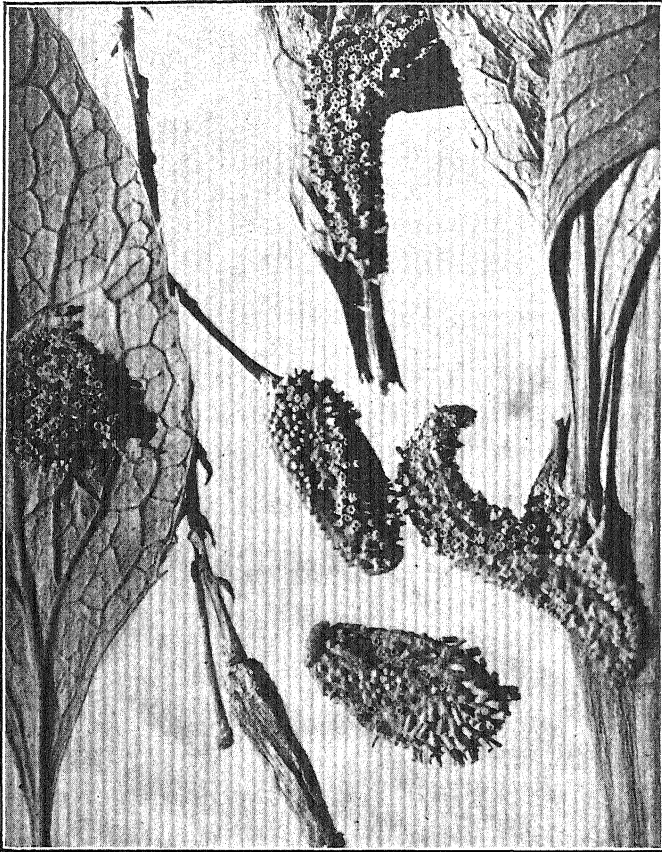


FIG. 126. — Aecia of stem-rust on barberry leaves and fruit.  $\times 3\frac{1}{2}$ . (After Gilbert, S. D. Agr. Coll. Ext. Cir. 33.)

extremely long pustules and the ragged, irregular appearance of the fringe of epidermis surrounding the open pustule.

On the barberry, yellowish or orange-colored spots appear on the leaves, young twigs and fruits. Close examination of these spots on the under side of the leaf will show a cluster of small round cups (aecia) (Fig. 126). Each aecium is about half a millimeter in diameter. On

the upper side of the spot a few very minute, dark-colored papillae (pycnia) may be seen. These are too small to be seen readily without the aid of a hand lens. Usually the leaf tissue within such areas is thickened considerably. These symptoms appear on the barberry soon after the leaves unfold in the spring (see under Life History).

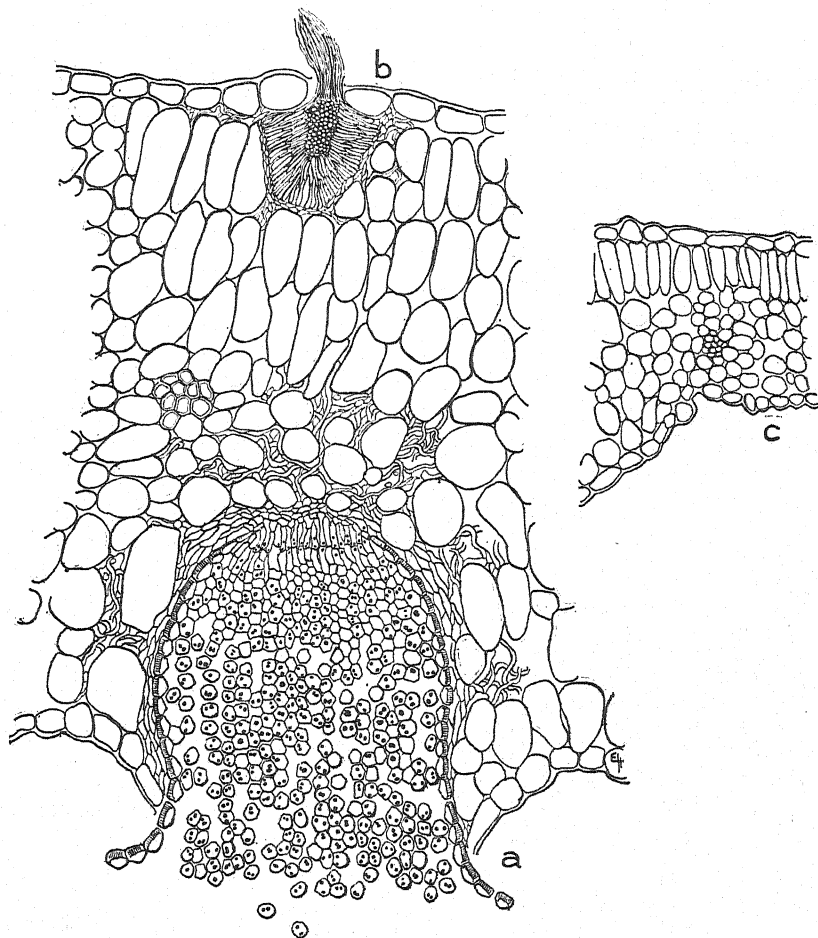


FIG. 127. — Section of aecium (a) and pycnium (b) of *puccinia graminis* on barberry leaf. (c) shows the normal thickness of the healthy leaf, in comparison with the hypertrophied effect in the diseased area.

**Morphology and life history of the rust fungus.** — This organism is a heteroecious eu-type rust fungus. By referring to the general discussion of rusts in a previous section it is readily seen that this fungus has all the

spore forms known among the rusts and that it completes its life cycle on two alternating hosts.

O. The pycnial stage borne on the barberry consists of flask-shaped structures, pycnia, which bear, on minute thread-like stalks converging toward the center of the opening within the pycnium, very small spore-like bodies, called pycniospores (Fig. 127 *b*).

I. The aecial stage, also borne on the barberry, consists of clusters of small cups, aecia, which bear columns of spores, aeciospores, within the cup (Fig. 127 *a*). The aeciospores are functional, being the means by which the rust organism is transmitted from the barberry bush back to the wheat or other grass host. These spores cannot infect barberry bushes. The aeciospores are mature in late spring or early summer and thus serve to infect the grain while in its green, growing condition.

II. The uredinal stage is the first fruiting or sporulating stage to appear on the grain or grass host after infection by aeciospores has occurred. Urediniospores (Fig. 128) are produced in large numbers in the uredinia. They are oblong, one-celled spores. These spores serve to spread the disease rapidly among the plants of the grain or grass hosts, but are unable to infect the barberry. They are sometimes spoken of as repeating spores because infection on grain or grass hosts can repeatedly occur from urediniospores.

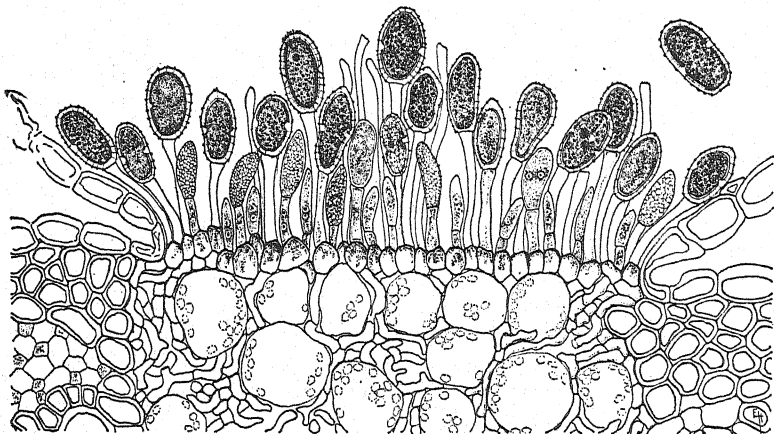


FIG. 128. — Section through a uredinium of *Puccinia graminis* on wheat stem, showing urediniospores in various stages of development.

III. The telial stage follows the uredinal stage, and usually appears as the host approaches maturity. Teliospores may appear in the same pustule with urediniospores or new pustules may appear bearing only teliospores (Fig. 129). The latter is likely to be the case later in the

season. The teliospores are two-celled spores with heavy, dark-colored walls. These spores winter over on dead straw or stubble and germinate in place the next spring when the barberry leaves are coming out. On germination the teliospore does not cause direct infection, but gives rise to a basidium on which are borne four basidiospores (Fig. 130). These basidiospores are capable of infecting only the barberry, never the grains or grasses. Unless these basidiospores find lodgment on the barberry they come to naught.

Thus it is seen that only one type of spore of the stem-rust can infect barberry, namely, the basidiospore, but that two kinds of spores can infect the grain or grass, namely aeciospores and urediniospores.

*Overwintering.* — One of the most important questions in connection with the life history of this rust is that of carrying the disease over winter.

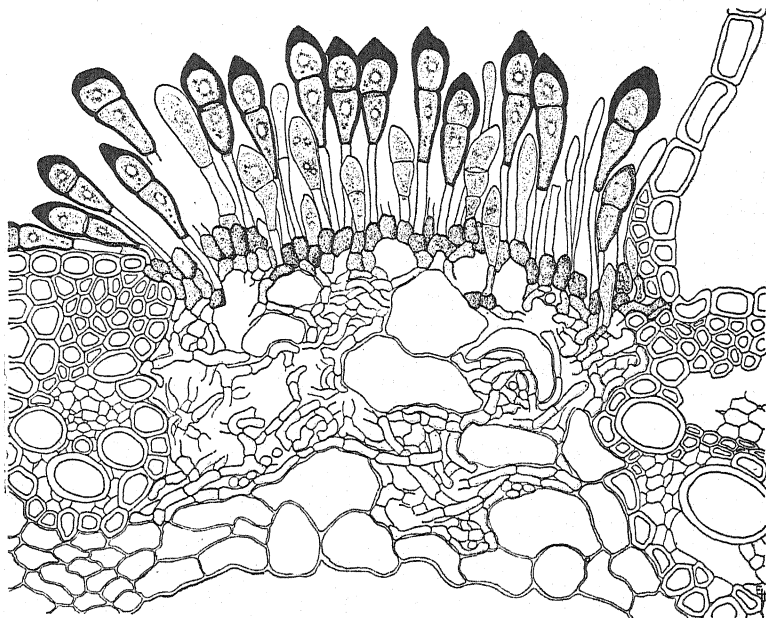


FIG. 129. — Section through a telium of *Puccinia graminis* on wheat stem, showing teliospores in various stages of development.

It has already been shown that teliospores overwinter and give rise the next spring to basidiospores, which infect barberry bushes if the latter are present. The question then naturally arises as to whether the barberry is necessary for the perpetuation of the rust from year to year. This leads us to ask if there might not be any other means of overwintering. Since urediniospores and aeciospores are the only ones capable

of infecting wheat, the only one left, if aeciospores from the barberry are eliminated, is the urediniospore. A great deal of research has been done to determine if urediniospores can overwinter and start the disease the following spring. The general opinion prevails at present, as a

result of these investigations, that in regions where the winters are severe not many urediniospores survive and not much rust would occur if there were no barberries. In milder climates, however, the rust is able to overwinter in the mycelial or urediniospore stage on living grains or grasses and thus is perpetuated from year to year without the barberry. There seems to be some evidence also that different strains of the rust may differ in their ability to overwinter without the barberry.

It has been claimed by many that the rust may overwinter either as spores or as mycelium in the seed. Hungerford (5) has shown quite conclusively that while both urediniospores and teliospores are sometimes found imbedded in seeds, the disease is not transmitted to the new crop in this manner.

*Dissemination.* — The wind is probably the only agent of any importance concerned in the dissemination of stem-rust spores, whether from barberry to grain, from grain to barberry, or from grain to grain or grass.

*Infection.* — Infection takes place best under warm humid conditions. Heavy dews, fogs, and gentle rains, when the temperature is fairly high, offer ideal conditions for infection and spread of the rust, which develops more slowly at low temperatures.

*Epidemiology.* — In the great wheat belt of the north central United States epidemics of stem-rust are not uncommon. Such outbreaks occurred during the years 1916, 1919, 1920, 1921, 1923 and

1925. Before the barberry eradication campaign was carried out it was customary to place the blame for such epidemics upon the barberry. However, this alternate host of the rust had been eradicated from large areas of the wheat states before the outbreak of 1925 which overspread much of the hard red spring-wheat region. Naturally this raised the question as to the source of inoculum in this outbreak.

There are three possible sources (23) of rust in this region: (a) ure-

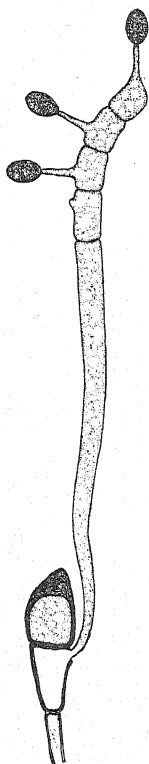


FIG. 130. — Germinating teliospore of *Puccinia graminis* showing basidium (pro-mycelium) and basidiospores (sporidia).



diniospores which may have overwintered here and there on fall-sown grains, volunteer grains, or wild grasses; (b) urediniospores blown northward by strong winds from Texas and Mexico where they may have overwintered in the milder climate; and (c) the common barberry bush. It is not possible at the present time completely to eliminate any one of these three sources. Urediniospores do not usually overwinter in this northerly climate but instances are known where a few have done so in favorable locations. It is, therefore, probable that primary infections occasionally occur from this source. It is almost impossible to locate every barberry bush no matter how thoroughly the eradication work is done. The bushes which escape detection or for some other reason are not eradicated undoubtedly may serve as foci for the spread of aeciospores to nearby grain fields. Lastly, there is the possibility of spores of southern origin being blown to the wheat fields of the northern states. It is also possible that the large areas of low pressure and cyclonic wind movements may bring spores from the eastern United States into the wheat belt of the upper Mississippi Valley. This question of wind-carried urediniospores needs further investigation and an attempt is being made at the present time to solve this question by trying to trap spores at high altitudes by means of airplanes.

In any discussion of epidemiology the climatic and weather conditions must be taken into consideration. No matter how abundant the source of inoculum may be, if conditions favorable for spore germination and infection do not obtain there is little danger of an outbreak. On the other hand the great cyclonic storm areas which sweep over the section east of the Rocky Mountains bring about exactly the conditions necessary to initiate an outbreak of stem-rust provided the spores are brought in with the storm winds or are already present from one of the other sources mentioned.

In spite of this evidence, however, there is no good reason to conclude that barberry eradication is not effective. The epidemic of 1925 was not as destructive as that of 1916 in spite of the fact that weather conditions were just as favorable for an epidemic in the former year as in the latter. The difference in severity can be attributed to the eradication of barberries.

**Control.** — In view of all the facts discussed above, it is evident that several different factors must be considered in determining possible control measures for stem-rust. Some of these items are: (a) the part played by the barberry; (b) the overwintering of the uredinial stage; (c) weed-grass hosts; (d) resistant varieties and biologic races; (e) soil management, fertilizers, etc.

*Barberry eradication.* — It has been shown conclusively that in the



more northern climates the common barberry (*Berberis vulgaris*) is a very real menace to the grain crops and its eradication is now required by law in all the more northern wheat-growing states of the Union. It has already been noted that certain countries of Europe have long ago legislated against the barberry. The common barberry is not a native of North America but was imported in the early days and used as an ornamental and in hedges. In many places it has escaped from cultivation. There are several species of native barberries or closely related plants in this country, but none of these have ever been shown to be dangerous to the grain crop, although some of them are slightly susceptible to the stem-rust. The Japanese barberry is immune. The harmful common barberry can be identified by its raceme-like clusters of flowers or fruits and by the fact that the spines are long and usually in groups of three at the bases of the leaves.

*The uredinial stage.* — In warmer climates, as the Gulf States and on the Pacific Coast, the stem-rust fungus seems capable of perpetuating itself indefinitely in the vegetative and uredinial stages, so that barberry eradication is not effective in regions where these conditions prevail.

*Grass hosts.* — Many wild grasses are capable of acting as weed hosts for the uredinial and telial stages. Such grasses may at times be instrumental in intensifying rust attacks since urediniospores from these grasses can infect the cereals. Some grasses which rust heavily are the wild ryes, the wild barleys, some of the wheat-grasses and some brome-grasses. Where practical the eradication of these weed grasses is advisable.

*Immune varieties.* — Some excellent varieties of wheat which show resistance to stem-rust (10) have been selected or bred. It should be kept in mind, however, that the black stem-rust fungus is composed of a large number of biologic strains and it is not likely that any one variety or pure line of wheat will be found which is immune to all races of the rust fungus (see under Hosts above and also Chapter XI). The problem of securing or developing varieties of wheat which will be resistant or immune to stem-rust, then, resolves itself into a local one, since suitable varieties which will be resistant to the particular strain or strains of rust fungus found in any particular region must be found. Much progress has already been made in this direction.

*Soil management, fertilizers, etc.* — Certain items in the cultural practices of wheat-growing may contribute to a reduction in losses due to rust. Air drainage, date of seeding and maturing of the crop and the use of fertilizers all play a part. On low-lying ground, dews and rains dry off more slowly than on higher ground having good air drainage, and thus a better opportunity is afforded for rust infection and spread

to occur. Early maturing grain has a greater chance of escaping rust infection than later maturing grain. Early seeding as well as the use of early-maturing varieties will contribute toward control by this means. The use of fertilizers may act in two indirect ways to lessen or increase the amount of rust (22). Certain fertilizers may hasten the maturity and others may delay the maturity of the crop, thus either decreasing or increasing the probability of severe rust infection. Again, any method of fertilizing which causes a fluctuation in the proportionate amounts of mechanical and chlorenchyma tissues has its effect upon the morphologic resistance to rust. The larger the proportion of mechanical tissue in the stem the more resistant it seems to be, and *vice versa*. In general it seems better to avoid excessive use of nitrogen fertilizers, and to use phosphates and potassium fertilizers where the soil needs them.

#### LABORATORY STUDY OF STEM-RUST

##### A. Symptoms.

1. *On grains and grasses.* — Examine rusted specimens of wheat or other grains and note the symptoms and signs of the disease. On what part of the plant are the rust sori located, stem, leaf sheath, or leaf blade? Are the sori elongated in transverse or longitudinal direction? Note the margins of the open pustules. Note the color of the sori in the two different stages of the rust. The red or rust-colored sori are the uredinal stage and the black sori represent the telial stage. Of what does the rust-colored mass or the black mass in the pustule consist? (See under B below.) Make a habit sketch of a rusted straw. Draw one sorus enlarged to show its shape, margin and contents. Use hand lens, dissecting microscope or binocular microscope in making the latter drawing. Examine shriveled grain from a badly rusted field. Do all of these grains show rust sori? Why should rust on the stem of the wheat plant cause the grain to shrivel?

2. *On barberry.* — Note the appearance of the rust spots on both sides of a barberry leaf. Use hand lens, dissecting or binocular microscope. On which side of the leaf are the aecia? The pycnia? Make a habit sketch of the leaf showing the clusters of aecia and pycnia. Draw one cluster of aecia enlarged.

##### B. Morphology of the fungus.

1. *The uredinal stage* (Red rust). — Examine a section through a uredinium or scrape off some urediniospores with a scalpel and mount them. Note shape, color, markings and content of the spores. Draw the section or individual spores. Examine and draw germinating spores.

2. *The telial stage* (Black rust). — Examine sections of telia or mount teliospores from the specimens available. Compare with the uredinal stage. Are the pustules similar? Do both kinds of spores ever occur in the same sorus? How many cells in a teliospore? In a urediniospore? Compare the two kinds of spores as to color, shape and surface markings. If germinating teliospores are available, examine and draw. If not, look up illustrations showing promycelium and basidiospores.

3. *The aecial and pycnial stages.* — Study sections of barberry leaf showing these stages. Draw a strip through the section showing both an aecium and a pycnium in their relation to the leaf tissue. Can you see mycelium between the leaf cells?

Does the mycelium extend entirely through the leaf between pycnium and aecium? Note the contents of both the aecium and the pycnium. How many nuclei in each aeciospore? What is the significance of this? Look up the question of sexuality in the rusts.

**C. Life Cycle.** — After gaining a thorough understanding of the complete life cycle of the stem-rust fungus from the preceding laboratory study and from reading the text and other references, construct a diagram to illustrate the life history.

**D. Notes.** — Write a complete description of the symptoms of the disease and the morphology of the causal fungus. After completing the assigned reading include in your notes an account of the life cycle of the fungus and recommendations for the control of the disease.

#### REVIEW QUESTIONS

1. Name and describe all the spore forms occurring in the life cycle of the stem-rust fungus.
2. Name the respective hosts on which the various spore forms occur.
3. Describe the symptoms and signs by which each stage may be recognized.
4. Is it possible for this rust to be perpetuated from year to year in any other way than through the stage on barberry?
5. In what section of the country especially is the barberry thought to be an important factor in perpetuating and spreading the rust?
6. What is a biologic race or strain?
7. What does the question of biologic races have to do with the problem of securing varieties of wheat immune to stem-rust?
8. How can epidemics of stem-rust in the north central United States be accounted for in the absence of barberry bushes?
9. What is the probability of stem-rust being perpetuated and disseminated on wheat seed? (Reference 5.)

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## White-pine Blister-rust

Caused by *Cronartium ribicola* Fischer

Probably no plant disease has aroused greater apprehension nor been the subject of more extended investigation in the United States than the white-pine blister-rust, the presence of which was first discovered

in this country in 1906. There are extensive and valuable stands of white pine in the United States, and in certain sections, notably in the New England States, reforestation with white pine is being practiced quite widely. Therefore, any disease which threatens the destruction of this important timber tree at once causes great concern on the part of those interested in the preservation of our timber resources. As the disease has gradually spread in this country, both Federal and State Agencies have given more and more attention to its study until at the present time a large number of men are engaged in the various phases of the problem of controlling the scourge and saving the white pines of the country.

**History and distribution.** — The original habitat of the white-pine blister-rust is not certainly known, but there seems to be strong evidence that it came originally from Asia and that its native host was *Pinus cembra*. The fungus is first mentioned in mycological literature by Dietrich (10) who reported it in 1856 from the western part of Russia. He found the telial stage on species of currants, *Ribes nigrum*, *R. rubrum* and *R. palmatum*, and named the fungus *Cronartium ribicola*, the name which it still retains. In 1861 it was found on white pine in Finland (23), and four years later it was reported from Germany (15). In 1869 it was found on black currants in Sweden. An outbreak occurred on white pines in Denmark in 1883. In 1888 Klebahn (14), by making inoculations, proved that the aecial form on pine and the telial and uredinial forms on *Ribes* are but different stages of the same fungus. Previous to this time the rust found on the white pines had been classified as a *Peridermium* and its connection with the currant rust was not known. Either the rust spread rapidly or else more searching observations were being made for early in the twentieth century reports show that the disease was present over a large part of northern Europe where the white pine, *Pinus strobus*, had been planted extensively. This pine is a native of North America and had been introduced into Europe about 1705. There has been a suggestion on the part of certain European investigators that the disease came originally from America but this has been entirely discredited. The probability is that the rust was endemic in Asia and parts of Europe on other five-needle pines but not in severe enough form to attract attention. Later when it came into contact with *Pinus strobus* the latter proved to be much more susceptible than the original host and hence the severe outbreaks on this species of pine.

The blister-rust was first found in America on *Ribes* at Geneva, New York, in 1906. There is evidence that it had been in this country several years before its discovery at that time and place. The stage on pine

was first noted in 1909. By 1916 the disease had been observed in the states of New Hampshire, Vermont, Massachusetts, Connecticut, New York, Pennsylvania, New Jersey, Ohio and Indiana. There is strong evidence that the disease came into this country on pine seedlings from European nurseries and it was undoubtedly introduced at several different points on different shipments of nursery stock brought into this country during the years 1898 to 1910. Later it has been found in Michigan, Wisconsin, Minnesota and Ontario, Canada. In 1921, blister-rust was discovered in the western part of British Columbia and in the state of Washington around Puget Sound. It had been introduced into British Columbia on white pine seedlings several years earlier, possibly as early as 1910. It is now spreading rapidly around the Puget Sound region and has been found also in the inland part of British Columbia. In 1925 the rust was found on *Ribes nigrum* and *R. bracteosum* in northwestern Oregon.

**Hosts.** — Since this is a heteroecious rust there are two types of hosts on which it completes its life history. The aecial host includes the five-needle pines, that is, the pines which have five needles\* in each leaf-fascicle. All species of the white pines thus far tested have proven susceptible, to the blister-rust fungus. In 1922 Spaulding (34) listed eleven species of pines known to be susceptible as follows: *Pinus koraiensis*, *P. cembra*, *P. flexilis*, *P. strobiformis*, *P. lambertiana*, *P. parviflora*, *P. peuce*, *P. excelsa*, *P. monticola*, *P. strobus* and *P. aristata*. In the United States the three important species of white pine, from the commercial standpoint, are the Eastern White Pine, *Pinus strobus*, the Western White Pine, *P. monticola*, and the Sugar Pine, *P. lambertiana*. All of these are very susceptible to the disease. The telial hosts include a very large number of species of currants and gooseberries, usually all considered in the genus *Ribes*. A great many species of these plants have been found naturally infested and a much larger number have been infected by artificial inoculation. Moir (23) lists over 40 species of *Ribes* infected with blister-rust which he saw in various herbaria of Europe. Evidently all these species were naturally infected. Spaulding (34) lists 13 species found naturally infected in the United States up to 1922. He further gives a compilation of species artificially infected by different workers in Europe and America which includes over 60 species. In general the cultivated species are found to be more susceptible than the wild forms although some wild species are very susceptible. The black garden currant, *Ribes nigrum*, is probably the most susceptible of all species.

**Economic importance.** — The greatest damage done by this disease

\* *Pinus arizonica* and *P. torreyana* are five-needle pines but not white pines.

results from its attacks on the pine trees rather than upon the currants and gooseberries. Both the eastern and western white pines and the sugar pine are severely injured when exposed to attack. The fungus girdles branches and trunks and kills trees outright within a few years after they are first invaded. In Europe where the white pine from eastern North America has been planted in large numbers there is now some question as to the advisability of planting this tree so extensively on account of its susceptibility to the blister-rust. In the United States where the white pines comprise large areas of our native forests the menace is very great. The danger is especially great where reforestation of cut-over areas is in progress since the young trees are easily killed by the blister-rust fungus and *Ribes* species are also much more abundant here than in old stands of timber. The standing white pine in the United States is valued at \$500,000,000 (8). In Canada there is said to be \$600,000,000 worth of white pine still standing. In the western United States there are large virgin stands of white pine in Idaho and of sugar pine in California and southern Oregon. These are menaced by the presence of the blister-rust in British Columbia, Washington and northwestern Oregon.

**Symptoms.** — On pines infection takes place through the leaves and the first symptoms appear as yellow spots on the needles. With a hand lens these spots can sometimes be detected within 25 days after inoculation. After a few months the spotting becomes more conspicuous. The infection spreads from the leaves to the stems but a variable time elapses before any symptoms appear on the stems. The first symptom on the stem is a slight fusiform swelling due to the thickening of the bark which becomes more or less spongy or corky. Some time after the invasion of the bark, six to nine months after infection according to Clinton (2, 3), the pycnial stage may appear. This consists of small blisters from which a sweet, yellowish, sticky fluid exudes. This ooze is filled with the minute, functionless pycniospores. Still later, 2 or 3 years after infection, the aecial stage appears. This consists of large, yellowish blisters which break through the bark at numerous places on the surface of the canker (Fig. 131). The aecial pustules are rounded to elongate and more or less irregular in outline. They range from one-eighth to one-half inch in diameter. The peridium is a delicate whitish membrane surrounding the orange colored mass of spores as it breaks through the outer bark. Later this membrane breaks and the spores escape leaving the empty depressions in the bark still partly or entirely lined with the peridial membrane. Later all signs of the fungus may disappear leaving only the roughened and fissured bark over the surface of the swollen cankers. The fungus spreads in the bark from



year to year thus enlarging the canker as long as the trunk or branch remains alive. Finally the branch or trunk is completely girdled and dies.



FIG. 131. — Aecia of blister-rust on white pine tree. (Courtesy U. S. Dept. of Agr.)

The symptoms on *Ribes* consist largely of the fruiting pustules of the uredinial and telial stages and might be considered signs rather than

symptoms in the restricted sense. The uredinia are minute yellow pustules while the mature telia are recognized by the hair-like telial columns which arise profusely over the leaf surface (Figs. 132, 133). Both stages usually occur on the under side of the leaf, but in some cases the fungus may fruit on the upper side of the leaf.

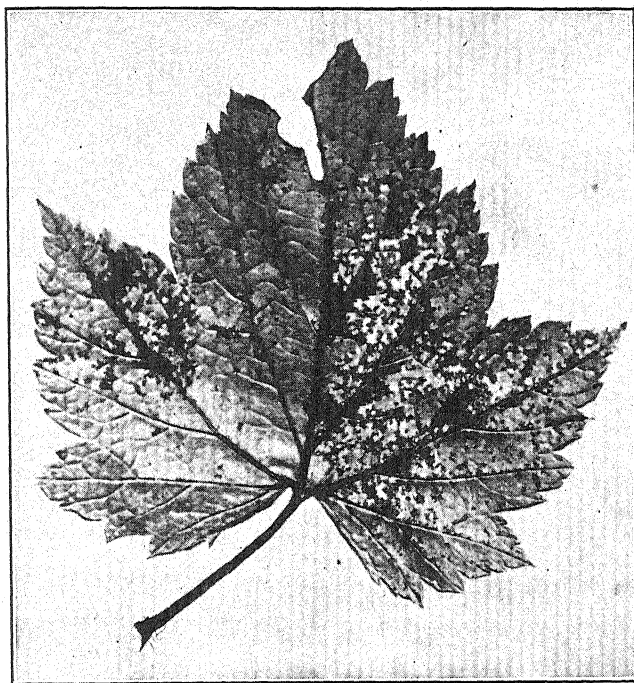


FIG. 132. — Telial stage of blister-rust on currant leaf. Natural size.

**Morphology and life cycle of the fungus.** — The white-pine blister-rust fungus is a heteroecious eu-type rust and hence all the known spore forms are present in its life cycle. The pycnial and aecial stages occur on pines, and the uredinial and telial stages are found on gooseberries and currants. The aeciospores can infect only gooseberries and currants, never pines so far as known, while the urediniospores can transmit the disease from currants and gooseberries to currants and gooseberries but never to pines. The only spore known to infect pines is the basidiospore, or sporidium, which is produced from the germinating teliospore. (See general discussion of rusts.) The aeciospores are borne in aecia as described under symptoms. These spores are ellipsoid to ovoid in shape and measure  $18-20 \times 22-23 \mu$ . The spore-wall is colorless and coarsely

verrucose. The urediniospores are borne in uredinia on the currant and gooseberry leaves. The uredinial pustules are minute, one-tenth to three-tenths millimeter in diameter, and bright yellow in color when in fresh condition. The urediniospores are ellipsoid to obovate,  $14-22 \times$

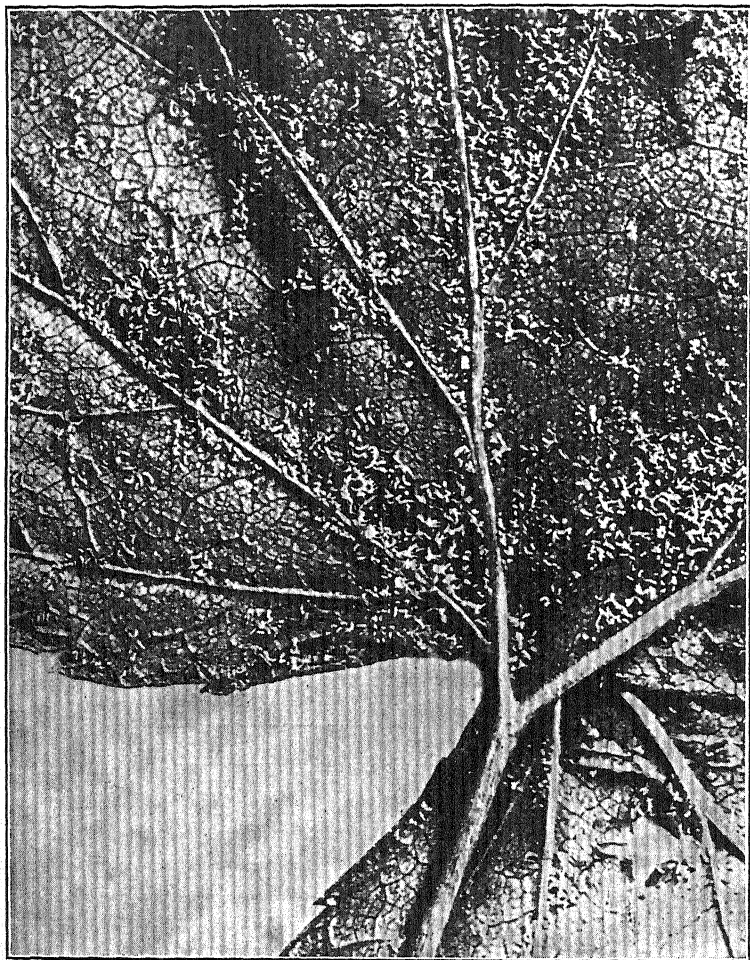


FIG. 133. — Telial stage of blister-rust on leaf of black currant, *Ribes nigrum*, enlarged to show the telial columns. (Photograph by W. H. Rankin.)

$19-35 \mu$  in size, and have colorless walls which are sparsely and sharply echinulate. The telia also occur on *Ribes* leaves and the teliospores are produced in columns which emerge from tiny pustules on the leaf and stand out like hairs over the leaf surface. These hairs or telial columns

may be as much as two millimeters long and one-eighth to one-sixth millimeter in diameter. They are at first yellow or reddish-brown and later become dark-brown in color. The telial columns are composed of cells each of which functions as a teliospore. The teliospores are oblong or cylindrical,  $8-12 \times 30-60 \mu$ , with smooth thick walls. The teliospores germinate by promycelia on which are produced basidiospores (sporidia) as is typical of the rusts in general (Fig. 134).

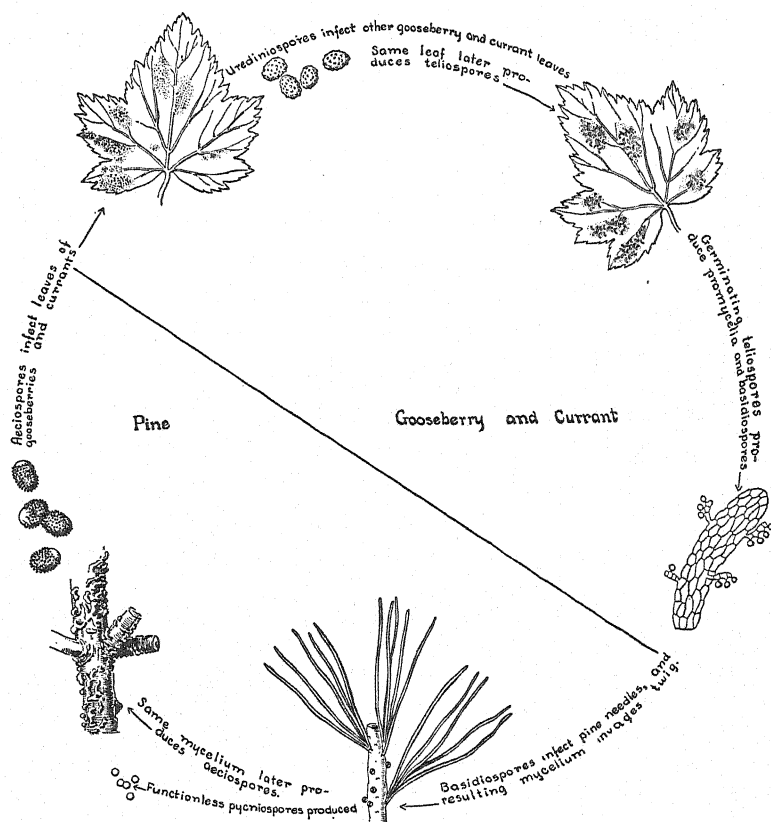


FIG. 134. — Diagrammatic representation of the life history cycle of *Cronartium ribicola*. For a discussion of the probable function of the pycniospores see under stem-rust of cereals.

The life cycle (Fig. 134) of *Cronartium ribicola* is difficult to trace and there has been a great difference of opinion among investigators as to the time required to complete the cycle. Teliospores may mature on *Ribes* at any time after June first and apparently infection on pine can take place at any time after that whenever the proper conditions for

spore germination prevail. Sporidia are produced within five or six hours after teliospores are placed under the right conditions for germination (43). The sporidia are short-lived and germinate immediately under the proper conditions of moisture. It is stated (42) that infection may occur on pine needles, under the most favorable conditions, in twelve to eighteen hours after viable sporidia reach the leaves. Apparently most infections of pines occur in late summer or autumn but it is possible for infections to take place at any time after teliospores are mature provided the right weather conditions prevail. According to Clinton and McCormick (3) the complete cycle of development on the pine is as follows: (a) During the first year, infection occurs on the leaves, producing only inconspicuous yellow spots. In rare cases the stem may be invaded before the end of the growing season. (b) During the spring and early summer of the second year the leaf-spots become more distinct and the stem is invaded by the fungus causing more or less swelling and discoloration. Sometimes pycnia may be produced the second season. (c) During the third season there is continued swelling of the diseased stem. Stunting of the leaves may occur. Pycnia develop during the summer. In those cases where pycnia develop during the second season there may be aecial formation the third spring. (d) In the fourth growing season aecia are commonly produced. In some cases, however, the first appearance of aecia may be delayed even beyond the fourth season after infection. The cycle as given by other investigators has varied somewhat from the above, especially as to the time involved, but considering the fact that in this schedule there is considerable variation as to the length of time required for both pycnial and aecial formation in different cases, it seems that the facts are probably about as given by Clinton and McCormick.

The aeciospores usually mature in the early spring and are carried by the wind or other agencies to the young leaves of currants or gooseberries. Mature urediniospores have been found as early as May 16, and teliospores sometimes mature in June. As many as seven generations of urediniospores have been recorded in a single season, the first generation originating from aeciospores and later generations coming from infections by urediniospores.

*Longevity and dissemination of aeciospores.* — The length of time during which aeciospores remain viable is, of course, open to question. Naturally the conditions under which the spores are kept will have much influence in shortening or lengthening this time. In one case (34, p. 39), aeciospores germinated in water after being kept dry in the laboratory for 157 days. In another case (34, p. 39), aeciospores lost their vitality within three weeks whether brought into the laboratory or left in the

field. The distance through which the disease can be spread by means of aeciospores has also occasioned a great deal of speculation. Most observations made on this point indicate that aeciospores may easily spread the disease from one to two miles. Circumstantial evidence indicates that aeciospores have been disseminated several miles and caused infection on *Ribes*. In fact there is strong evidence in the Pacific Northwest that aeciospores have caused infection at distances of over a hundred miles from their source. Experiments with spore traps indicate that the wind may carry aeciospores many miles from infected pines and that the spores remain viable during transit and are able to infect *Ribes* leaves at their destination. Aeciospores may be disseminated by wind, insects and on nursery stock.

*Longevity and dissemination of urediniospores.* — The longevity of urediniospores likewise varies with conditions. Spores kept out of doors, but protected from rain, have remained viable for 100 days. Under normal weather conditions with occasional rain, the urediniospores seem to have a much shorter life in general. Spores kept in the laboratory and well protected sometimes remain viable for more than 100 days. Spaulding (34) in summarizing the evidence to 1922 states that in various experiments urediniospores have remained viable for variable periods of time ranging from 7 to 270 days. In a later experiment, Spaulding and Gravatt (35) found urediniospores remaining viable for a period of 59 days. These results are indefinite and inconclusive, and the subject needs further study. The distance through which urediniospores are disseminated in nature is also a matter of conjecture. In general, the disease does not seem to scatter rapidly and through long distances from *Ribes* to *Ribes*. The distance through which viable urediniospores are disseminated by the wind varies from a few feet up to a few hundred yards according to all available evidence. In an open location spores have been carried half a mile but this seems to be unusual. At any rate they serve to bring about intensification of the disease on *Ribes* within narrow limits. How far the spores may be carried by insects or birds is not known.

*Longevity of teliospores and dissemination of sporidia.* — The latest information on the longevity of teliospores (35) indicates that they may retain their viability for at least 80 or 90 days in some cases. Dissemination from *Ribes* to pines occurs only by means of the sporidia from germinating teliospores. The sporidia are the most delicate and short-lived of any of the spore forms of the rusts. Various experiments have shown that sporidia will not survive drying for more than a day or two. There is no evidence that pines may be infected by sporidia at very great distances from currants and gooseberries. There is evidence

(28) that sporidia from black currants may infect pines at a distance of 600 to 900 feet from their source, while sporidia from other species of *Ribes* showed no evidence of infecting pines beyond 200 feet. The limit of spread from *Ribes* to pine, therefore, seems to be confined to distances of only a few hundred yards.

*Overwintering.* — Since the mycelium is perennial in pines for several years this is one sure means of hibernation. The question has many times arisen as to whether the fungus can overwinter also on currants and gooseberries. Considerable has been written on this subject and opinion seems to be about equally divided. The evidence is mainly circumstantial, being based upon field observations of outbreaks of rust on *Ribes* in the presence or absence of aecial infections on pine. In the absence of any definite data as to how far the aeciospores can travel from pine and still cause infection it would seem that it is not safe to conclude that overwintering occurs on *Ribes* simply because an outbreak of rust occurs on this host at a distance of a mile or two from any known infected pines. On the other hand there seems to be some ground for believing that the rust fungus may overwinter on *Ribes* leaves in certain cases under favorable conditions.

*Dissemination.* — The blister-rust fungus may be disseminated in several ways. All the spore types are undoubtedly disseminated very largely by the wind. Insects have also been incriminated. They apparently disseminate both aeciospores and urediniospores to a limited extent. Birds may also possibly aid in a limited way. Sporidia are probably disseminated almost entirely by wind. Man is an important agent when he transports diseased pine seedlings. This is undoubtedly the chief if not the only means by which the fungus makes long jumps of several hundred miles.

*Control.* — Based upon the known facts in the dissemination and behavior of the disease, the chief methods of control may be stated as follows: (a) by removal of the alternate host; (b) by quarantine regulations; (c) by eradication of isolated centers of infection; and (d) by sanitation.

In removing the alternate host the first question to be met, of course, is as to which host is the most important from the economic standpoint in the locality under consideration. There are certain sections of the country where currants and gooseberries are commercially of much more value than any stray white pines that might be in the neighborhood. But for the most part, in America especially, the problem is one of saving the enormously valuable stands of white pines. Here the problem becomes one of eradicating the *Ribes* species in the immediate neighborhood of the pines. It will be remembered that the sporidia will carry



the disease only a few hundred feet from *Ribes* to pine. Since both wild and cultivated species of currants and gooseberries must be removed this might at first thought be considered impractical but it has been demonstrated experimentally on a large scale in New England and other eastern states that it can be done. Several hundreds of thousands of acres of pine timber land have been cleared of *Ribes* bushes at an average cost of considerably less than a dollar per acre. In 1919 in New England the cost was finally reduced to the insignificant sum of 24 cents per acre. In some cases it may be feasible to pasture the bushes closely by goats or sheep. Of course there are difficulties to be met in this method of control. In densely wooded and rugged areas it is probably not possible to find and eradicate all wild *Ribes* bushes. However, it has been found that a few small bushes more or less covered by other vegetation are not much of a menace because they rarely become infected and in case they do become infected they produce few spores and these are not readily disseminated.

Quarantine regulations are now in force against shipment of nursery stock likely to carry the disease from infected zones into territory where the disease does not as yet exist. It is probable that if a strict quarantine had been in force against European shipments of white pine nursery stock at the time the rust was introduced into this country, we should still be free from this scourge. If a new center of infection is discovered in time it may be possible to stamp out the disease by eradication of all diseased plants, both pines and *Ribes*. All infested plants should be destroyed as a matter of precaution since it has been shown that felled diseased pine trees may produce aeciospores for a time after being cut down, especially if in a shaded and moist position. In case of ornamental pines diseased branches and cankers may be cut out and the disease eradicated in that way.

#### LABORATORY STUDY OF BLISTER-RUST

**A. Symptoms.** — Examine specimens of pine and also of currants or gooseberries showing the various stages of the rust.

*On pine.* — Observe both pycnial and aecial stages, also needles showing the first symptoms of infection. Read the text and find out at what season of the year and on what part of the pine infection occurs. How long after infection occurs before the pycnial stage appears? The aecial stage? What effect does the fungus have on the bark? On the wood? Account for the disappearance of the bark on diseased areas in some cases. Make drawings to illustrate all symptoms observed. Describe in your notes.

*On Ribes.* — Examine *Ribes* leaves showing both uredinial and telial stages. Note the color, shape and size of the sori in both stages. On which surface of the leaf do these sori occur? Are there any symptoms of the disease on the opposite surface of the leaf?



**B. Morphology and life cycle of the fungus.** — Examine all spore stages with the microscope. Compare the urediniospores with the aeciospores as to size, shape, color and wall characteristics. Draw. If sections of *Ribes* leaves are available draw a sectional view of a uredinium. Examine the telial columns under low and high power. Of what is a telial horn composed? Note the general characteristics of a single teliospore. Draw a telial horn showing its cell structure. If germinating teliospores are available draw promycelium and basidiospores.

*Life cycle.* — What is the function of each spore form in the cycle? Describe the complete life cycle step by step. In what stage does the fungus overwinter? How disseminated from pine to *Ribes*? From *Ribes* to pine? From *Ribes* to *Ribes*?

**C. Notes.** — Write full notes giving a complete account of this disease in all details of symptoms, life history and control, also giving brief attention to history, geographical distribution, hosts and economic importance.

#### REVIEW QUESTIONS

1. From what country was blister-rust introduced into the United States? On what? When?
2. What is the present distribution of the rust in North America?
3. Describe the complete life history of the blister-rust fungus.
4. What is the nature of the damage done to pines?
5. What stage of the fungus is found on pines?
6. Describe the appearance of the two stages found on *Ribes*. Which spore intensifies the disease on *Ribes*?
7. Which spore stage is apparently able to disseminate the disease over long distances?
8. Which spore is able to survive only a short time and can transmit the disease only over very short distances? What has this fact to do with practical control measures?
9. What two control measures have been used to stop the spread of this disease? Which one is now being practiced successfully in connection with the white-pine plantings in the New England States and is being tried out experimentally in the native forests of certain western states?

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### Apple-rust

Caused by *Gymnosporangium juniperi-virginianae* Schw.

This is a common disease of apples in the eastern half of the United States wherever the juniper (*Juniperus virginiana*), commonly known as red cedar, occurs. The fungus is a heteroecious rust, having its telial stage on the cedar and its aecial and pycnial stages on the apple. No uredinal stage occurs. On the apple both fruits and leaves (Fig. 135) are affected. On the juniper the disease is characterized by the production of the large galls which are particularly conspicuous in the spring when the long, yellow, gelatinous telial horns cover the galls (Fig. 136). The teliospores in these horns germinate and produce basidiospores as is characteristic of teliospores in general. The basidiospores infect the young apple leaves and fruits as soon as they appear in the spring. The most successful means of control consist in eradicating all cedar trees within approximately a mile of the apple orchard. For further information on this disease see the following references.

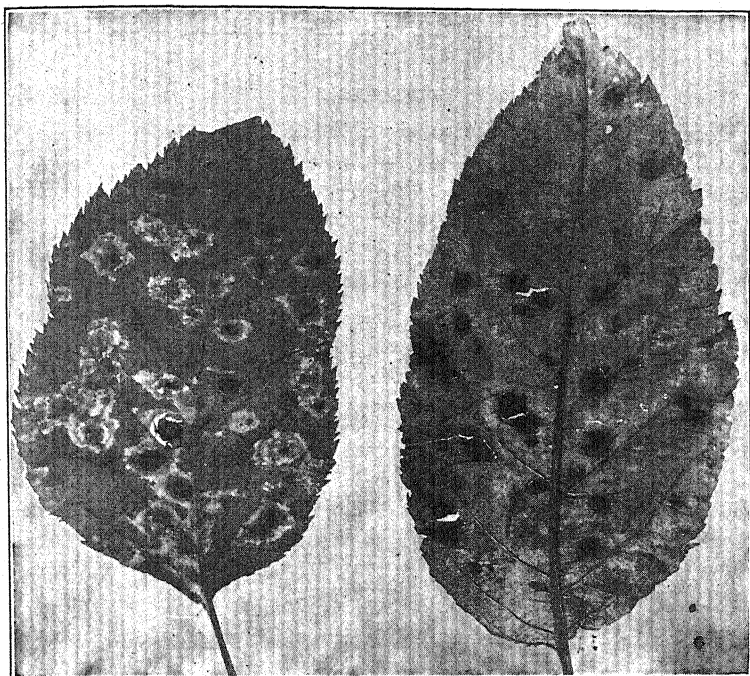


FIG. 135. — Aecial stage of the cedar rust on apple leaves. Right, aecia on under side of leaf. Left, pycnia on upper side of leaf.

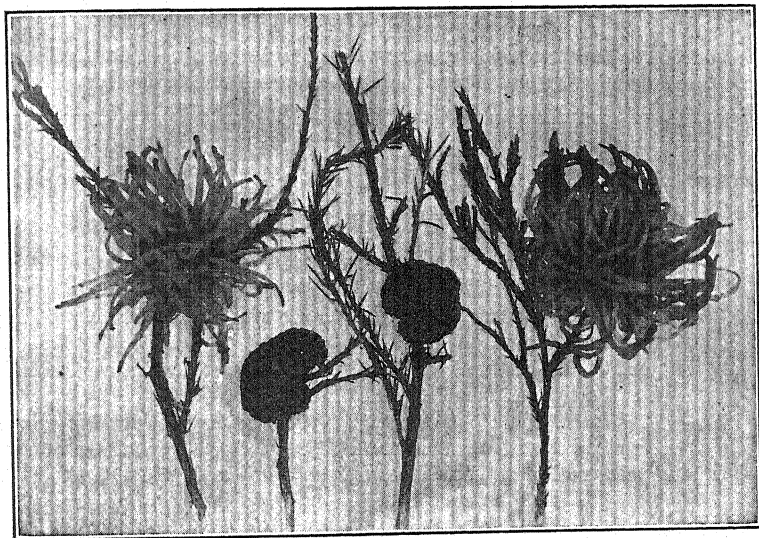


FIG. 136. — Galls produced by *Gymnosporangium juniperi-virginianae* on the eastern red cedar or juniper tree. Figures at right and left show the long gelatinous telial horns as they appear when the galls are in full "bloom" in the spring. Center, old galls after the telia have disappeared.

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In addition to the cereal rusts and the white-pine blister-rust previously discussed, there are many other species of rust fungi which attack economic plants as well as plants of little or no economic importance. A few of the economic species are mentioned below with one or more references pertaining to them.

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## Rhizoctonia of Potatoes

Caused by *Corticium vagum* B. and C.

This disease, which has been known under various names, including black-scurf, black-speck, black-speck scab, russet-scab, stem-rot, foot-rot, brown-stem, canker, collar-rot, rosette and Rhizoctonia, is one of the most common and widespread diseases of the potato in the United States. The causal organism, *Corticium vagum* B. and C., attacks other plants besides potatoes, causing various symptoms to which such other names as damping-off, seedling-rot and sore-shin have been applied.

**Historical.** — The particular species of fungus causing this disease was first described in Europe by Kühn in 1858 and named *Rhizoctonia solani*. In America, a fungus, apparently identical with the potato *Rhizoctonia*, was reported by Pammel (10) in 1891 as causing a beet root-rot in Iowa. In 1892, Atkinson (1) published the results of studies on a cotton disease in Alabama which was caused by a fungus resembling the *Rhizoctonia* fungus. In 1901 Duggar and Stewart (4) reported the *Rhizoctonia* disease on beets, beans, lettuce, radishes, cucumbers and other plants. In 1902, Rolfs in Colorado reported a serious potato disease caused by this fungus. Rolfs (13) later, in 1903, found the basidiospores of the fungus for the first time. From this time on plant pathologists in various parts of the country rapidly accumulated evidence that this fungus is the cause of a serious disease of potatoes as well as of various other plants both wild and cultivated.

**Geographic distribution.** — This disease occurs throughout the potato-growing sections of the United States and Canada. *Rhizoctonia* is also known to occur on potato or other plants in South America and the West Indies, in various countries of Europe, in Australia and in India.

**Hosts.** — We are here discussing this disease especially as it occurs on the potato, but it should be remembered that *Corticium vagum* attacks a very large number of species of plants distributed in several families. Peltier (11) observed the fungus on seventy-five different species of plants, including field, vegetable, and ornamental crops, and some weeds. He further states that the total number of species reported as more or less susceptible is about one hundred and sixty-five. Among the families to which the more susceptible species belong may be mentioned the pig weeds (*Amaranthaceae*), the pink family (*Caryophyllaceae*), the mustard family (*Cruciferae*), the legumes (*Leguminosae*), the nightshade family (*Solanaceae*) and the *Compositae*. These are mostly dicotyledons but some monocotyledons are susceptible as well as several gymnosperms and equisetum.

**Economic importance.** — *Rhizoctonia* on potatoes is undoubtedly one of the most generally prevalent diseases attacking this crop. The impression has been current for a good many years that, considering the country as a whole, *Rhizoctonia* takes a heavier toll from the potato crop than any other disease. More recently, however, in the opinion of some investigators, the virus diseases, such as the various types of mosaic and others, are outranking this disease in importance and it is thought that some of the losses formerly attributed to *Rhizoctonia* may have been due to mosaic and kindred troubles.

The Office of The Plant Disease Survey of the Bureau of Plant Indus-



try, United States Department of Agriculture, estimated the reduction in yield due to potato Rhizoctonia in the United States at nearly 17,000,000 bu. for the year 1922, approximately 13,000,000 bu. in 1923, and about 15,000,000 bu. in 1924.

**Symptoms.** — Considered in its broadest sense as a disease of a wide range of hosts, Rhizoctonia produces a variety of symptoms. These include damping-off, stem-rot, root-rot, scab and secondary effects such as rosette and leaf-roll. With special reference to the potato the various symptoms and signs are (a) stem, root and stolon lesions or cankers, (b) aërial tubers, (c) little potatoes, (d) black-speck or scurf (sclerotia), (e) Rhizoctonia scab, (f) tuber-pits, (g) tuber-rot, (h) root-rot, (i) leaf-roll, and (j) rosette.

*Stem, stolon and root cankers.* — Lesions may occur on any of the underground parts of the plant. Frequently the young sprouts are "burned" off before they emerge from the ground. Cankers may occur on the main stem of more mature plants just at or below the ground surface. These are in the form of dead, brown areas in the cortex and may partially or entirely girdle the stem (Fig. 137). Sometimes these lesions penetrate only to the cam-



FIG. 137. — Rhizoctonia cankers on stem of potato.  
(After McKay, Ore. Agr. Exp. Sta. Cir. 24.)



bium, leaving the xylem uninjured. Again, especially in the later stages of the disease, the wood of the stem may be penetrated to such an extent that wilting results or the stem may even be completely rotted. The stolons on which the tubers are borne and the roots also may show lesions, girdling or rotting off similar to the symptoms on the main stem.

*Aërial tubers.* — A striking symptom sometimes evident in plants affected with this disease is the formation of small tubers from swollen

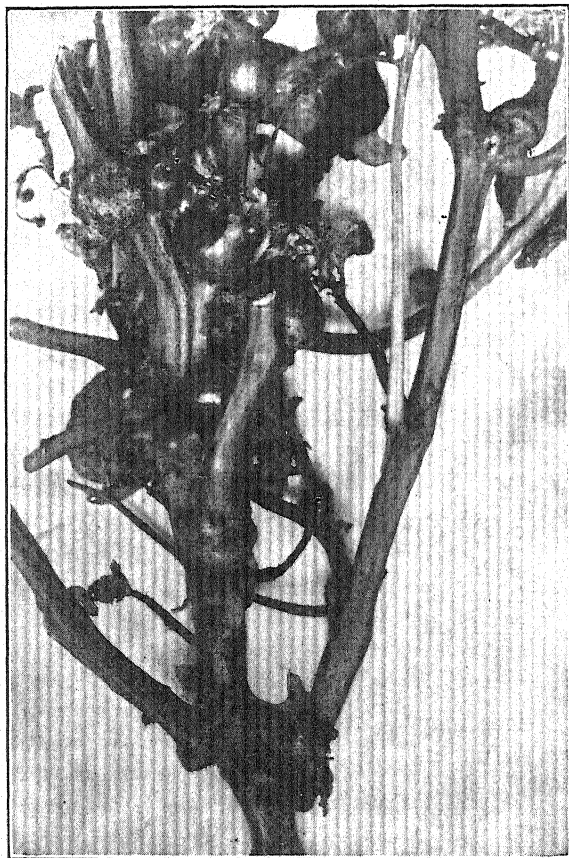


FIG. 138. — Aërial tubers produced as a result of girdling of the stem and stolons by the *Rhizoctonia* fungus. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest and Hort. Rept., 1911-12.)

branches in the axils of leaves above ground (Fig. 138). Their growth is stimulated as a result of the severing of the phloem channels by the cankers lower down on the stem or on the stolons leading to the normal tubers.

*Little potatoes.* — Sometimes a handful of small potatoes the size of marbles, or else a reduced number of tubers instead of the normal yield, may be found in a hill. These symptoms are due to conditions similar to those causing aërial tubers, namely, the cutting of the phloem cells in the stems and stolons which normally carry the elaborated food from the leaves where it is manufactured to the underground tubers where it is normally stored. If this girdling occurs while the tubers are young, naturally they will never grow to large size for lack of food.

*Leaf-roll and rosette.* — Potato plants which are attacked by the *Rhizoctonia* fungus on the underground parts sometimes show leaf-roll and rosette symptoms. *Rhizoctonia* leaf-roll may be difficult to distinguish from true leaf-roll, a virus disease, or from certain physiological symptoms due to water shortage. Selby described rosette as a symptom of *Rhizoctonia* and various subsequent writers have accepted rosette as a typical *Rhizoctonia* symptom. Orton (9) and Dana (3) suggest that rosette as well as leaf-roll and other secondary symptoms may also be the result of virus or possibly "physiological" diseases and not necessarily symptoms of *Rhizoctonia* only.

Other general symptoms appearing on the above-ground parts are wilting and general yellowing of the foliage. These symptoms may appear on plants that are severely affected by stem lesions or root-rot.

*Sclerotia.* — One of the most prevalent and easily recognized signs of the disease is the presence of sclerotia on the surface of the tubers.

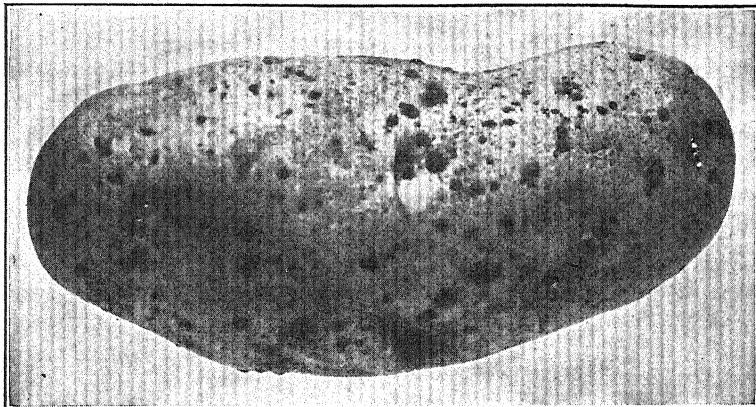


FIG. 139. — Potato tuber showing sclerotia of the *Rhizoctonia* fungus scattered over the surface. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest and Hort. Rept., 1911-12.)

These structures are small, black or brown, crust-like masses of dormant mycelium, varying in size from small specks scarcely visible to the naked

eye up to masses several millimeters in diameter. Sclerotia are sometimes produced on the roots and lower parts of stems but are most abundant on the tubers. The presence of sclerotia on the tubers has given rise to the terms "black-scurf," "black-speck" and "black-speck scab" (Fig. 139).

*Rhizoctonia scab.* — In contrast with the black-scurf described above, which does not penetrate the skin of the tuber, there sometimes develops a type of injury to the surface of the tuber which in its severe form greatly resembles the common scab caused by *Actinomyces scabies* (Thaxt.) Gussow. This scab type of symptom, sometimes called "russet scab," varies from a slight roughening of the skin to very scabby effects and may cover only a small area or may spread over the entire surface of the tuber.

*Tuber-rot and tuber-pit.* — A type of stem-end rot in tubers has been attributed to *Rhizoctonia* only occasionally (3, 15). The *Rhizoctonia* fungus is said to cause a characteristic pitting of tubers (8, 12). The typical symptom consists of an oval pit from 3 to 5 millimeters in diameter. This trouble apparently starts in the lenticels.

*Root-rot.* — Roots of diseased plants may be affected in several ways. The root cortex may be corroded in the same manner as that of stems, thus partially or entirely girdling the roots. Sometimes roots may be entirely rotted. Again the girdling of the stem may prevent a proper supply of elaborated food from reaching the roots with resultant stunting effects similar to those suffered by young tubers when their food supply is cut off.

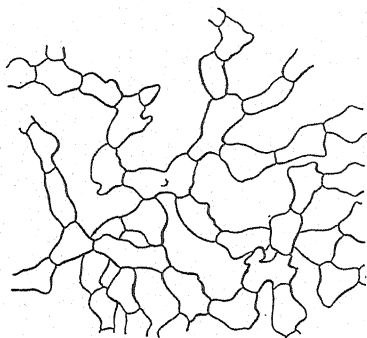


FIG. 140. — This drawing illustrates the cell structure of a fragment of a sclerotium of *Corticium vagum*.

**Morphology of the fungus.** — The fungus, *Corticium vagum*, shows three different phases in its complete life cycle, namely, the vegetative mycelium, sclerotia and a fruiting (basidiospore) stage.

*Vegetative mycelium.* — This consists of coarse hyphae with septa widely spaced. The hyphae branch freely, the branches tending to grow generally in a direction nearly parallel to the parent hypha. These hyphae turn dark in color on the surface of affected parts. The vegetative my-

celium may grow saprophytically in the soil or parasitically in or on the underground parts of plants.

*Sclerotia.* — The general appearance of these structures has been described above under Symptoms. Sclerotia are formed by the massing

together of tufts of hyphae consisting of much-branched, short, irregular, swollen cells (Fig. 140) on the surface of affected hosts, especially on potato tubers. Sclerotia have the ability to survive long unfavorable periods in a dormant period and initiate growth again upon the return of favorable growth conditions. The swollen cells in the sclerotium evidently contain stored food and any one of these cells may resume growth by putting out a hypha in much the same manner that a spore sends out a germ tube.

*Basidiospores.* — Up until 1903 no spore stage was known for this fungus; hence it was placed in the form genus, *Rhizoctonia*, which includes sterile fungi infesting the roots of plants. The name of this form genus still persists as one of the common names of the disease. In 1903 Rolfs discovered a spore stage, and this of course necessitated a revision of the name of the fungus.

The spore stage occurs on the stems of affected plants, a short distance above the ground. It consists of a weft or felt of whitish, buff or creamcolored mycelium, which surrounds the stem (Fig. 141). In this weft of mycelium, typical basidia with 4 to 6 sterigmata appear and basidiospores are produced (Fig. 142).

*Life cycle.* — As has been stated, the mycelium of *Corticium vagum* is capable of extended saprophytic life in the soil. The sclerotia constitute a dormant stage capable of carrying the fungus through long resting periods, easily surviving the winter season, either on potato tubers or in the soil. The swollen cells in the sclerotium act as chlamydospores, germinating under proper conditions and starting a new growth of vegetative mycelium which is



FIG. 141. — The fruiting stage of *Corticium vagum*. The whitish film of mycelium on the stem produces basidia and basidiospores. See Fig. 142. (After McKay, Ore. Agr. Exp. Sta. Cir. 24.)

capable of infecting growing plants. The basidiospores are capable of germinating and starting a saprophytic growth of mycelium, but it is a question whether they are able to cause direct infection on living plants. It is probable that the sclerotia are chiefly concerned in perpetuating the disease over winter and causing new infections on succeeding crops.

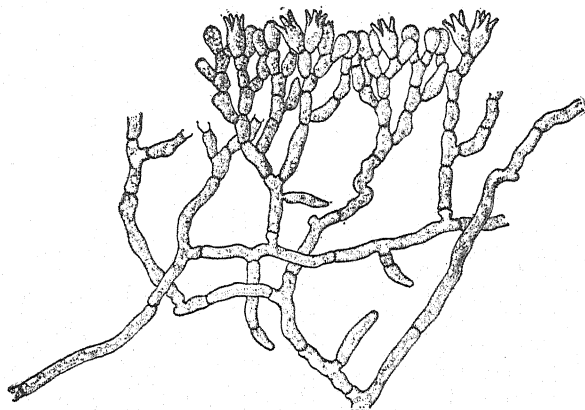


FIG. 142. — A few hyphae and basidia from the fruiting layer of *Corticium vagum* shown in Fig. 141.

**Control.** — With particular reference to the potato, there are chiefly two things that must be considered when devising control measures for the Rhizoctonia disease. First, the sclerotia are commonly present on the tubers. In many localities it is difficult to find seed potatoes free from sclerotia. Tubers which, on casual examination, seem to be free from the fungus may prove to carry small sclerotia or mycelium, if examined more closely. Second, this fungus attacks many other plants besides potatoes, and since sclerotia and saprophytic mycelium remain in the soil and on plant debris from year to year, it is difficult, and usually impossible, to find soil entirely free from the Rhizoctonia fungus. The recommendations, therefore, call for two chief items of control, (a) the use of clean seed and (b) crop rotation.

*Clean seed.* — Since it is not easy to select absolutely clean seed potatoes because of the difficulty of detecting small amounts of the disease on tubers, it becomes necessary to disinfect the seed tubers. One of the most effective fungicides for this purpose is bichloride of mercury used as follows: Dissolve 4 ounces of the bichloride of mercury (corrosive sublimate) in 30 gallons of water in a wooden, cement or earthenware vessel. Mercuric chloride corrodes metals. Treat the tubers in this solution for about one and one-half to two hours and dry

before planting. This solution decreases in strength with use, because the mercuric bichloride reacts chemically with organic matter and is thus taken out of solution. This can be corrected by adding one-half ounce of the chemical for every 4 bushels of potatoes treated. Corrosive sublimate is very poisonous and treated potatoes should not be eaten by man or animals.

More recently hot formaldehyde has been successfully substituted for mercuric chloride in the treatment of seed potatoes for Rhizoctonia. Its chief advantage is that large quantities of potatoes can be treated in much shorter time than by the corrosive sublimate method. The procedure consists essentially in dipping the potatoes, in sacks, in a solution of formaldehyde, 1 pint to 15 gallons of water, held at a temperature of 124° to 126° F., for 3 to 4 minutes. Potatoes thus treated are not poisonous and the solution does not corrode metals.

*Rotation.* — Crop rotation is to be recommended in the control of Rhizoctonia for although it is practically impossible, for reasons previously discussed, to rid any particular field of the fungus entirely, yet the continued cropping of the same ground with potatoes results often in a great increase of Rhizoctonia damage while a crop rotation of several years including a grain crop usually results in reducing the amount of this and other potato pathogens in the soil. Rotation and seed treatment combined afford the grower a profitable reduction in losses from this disease.

**Rhizoctonia in other crops.** — There is no intention of discussing Rhizoctonia diseases of other plants in detail. There are too many of them to treat here. One of the most important troubles attributed to this fungus, in addition to the potato disease, is the damping-off which it causes in greenhouses, seed beds and cutting benches. For a more detailed discussion of this type of trouble see page 226. For a discussion of Rhizoctonia disease in various other crops see some of the references following this exercise.

#### LABORATORY STUDY OF RHIZOCTONIA

**Symptoms.** — Examine diseased specimens of tubers, roots, stolons and stems of the potato and note the symptoms exhibited. Can you find all the symptoms described in the text? Try describing the symptoms in your own words. If specimens of plants other than potato, showing the effects of Rhizoctonia disease, are available, compare them with the potato specimens. Make drawings showing the various symptoms on potatoes.

**Morphology of the fungus.** — Use prepared sections of the sclerotia or make your own mounts by teasing out a small bit of a sclerotium on a slide. Examine microscopically. Describe the structure of the sclerotium. Draw typical cells or hyphae.

Likewise make mounts of the fruiting weft of mycelium from a stem. Draw hyphae, basidia and basidiospores.

**Cultures.** — If pure cultures of the fungus are available, describe the cultural characteristics of the growth on any media available. Mount mycelium from the cultures and compare with mounts previously studied.

**Inoculations.** — If greenhouse space is available and time and other conditions make it feasible, it will be of interest to start potatoes growing, inoculate them with the *Rhizoctonia* fungus and watch the appearance of the various symptoms of the disease.

**Notes.** — Write a complete account of this disease, following the outline on page 152 in so far as it is adapted to this disease. Get information on the various points in the outline from any source available, including laboratory studies, text and the references following.

#### REVIEW QUESTIONS

1. Explain the causal relations in the formation of aërial tubers.
2. What part do the spores play in the life cycle of *Corticium vagum*?
3. Why treat seed potatoes if the fungus lives over in the field anyway?
4. Why will not crop rotation completely rid a soil of this fungus?
5. Why not rely upon seed selection rather than seed disinfection in combating this disease?
6. Name some crops other than potatoes attacked by the same fungus. (See References 1, 2, 7, 10, 11.)
7. Discuss *Corticium vagum* as a damping-off fungus. (See References 1, 2.)
8. Compare the violet root-rot fungus, *Rhizoctonia crocorum*, with the common *Rhizoctonia* fungus. (See Reference 5.)

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### Wood-decay

Caused by various wood-rotting fungi, chiefly Basidiomycetes

The rotting of wood under various conditions is a phenomenon with which everyone is more or less familiar. In the forest the dead or fallen trees usually exhibit various stages of decay and the shelf or bracket fungi on dead logs and stumps are a familiar sight to anyone who has ever been in a forest. Fruit trees and shade trees are subject to decay and it is not unusual to see old shade or street trees with large rotten areas or hollow cavities in the trunks. In the orchard on the older trees one frequently sees large branches broken off because of the weakening effect of heart-rotting fungi. The decay of fence posts, telephone poles, railroad ties, pilings and bridge timbers are instances of serious damage resulting from attacks of fungi.

Wood-rotting fungi of many species occur all over the world wherever trees are found. Not all species are cosmopolitan but some of the common forms of the north temperate zone have a remarkably wide distribution. Many of our most abundant and destructive species occur in both Europe and America. Many of the species of the tropical zone, however, are not found in the more temperate climates and likewise some of the familiar species of the northern United States and Europe do not occur in the tropics.

**Host relationships.** — There is a great variation among the different species of wood-rotting fungi in their ability to attack different species of trees. Some species are confined to one or a very few kinds of trees while others are able to invade a very wide range of tree species. Probably the most noticeable host barrier is that existing between the Gymnosperms and the Angiosperms. There are many species of fungi which are confined in their attacks to the conifers, and many other species which live on the non-coniferous trees but not on the conifers. On the other hand there are a considerable number which can live on either type of tree. There are several species which are usually found on coniferous trees but occasionally occur on a few non-coniferous trees; for example,



the red-belt fungus, *Fomes pinicola*, and the pink-bracket fungus, *Trametes subrosea*, ordinarily are found in abundance on conifers but occasionally they have been found on non-conifers also. The pink-bracket fungus is especially prevalent on peach and prune trees in Oregon. The sulfur fungus, *Polyporus sulphureus*, is most commonly

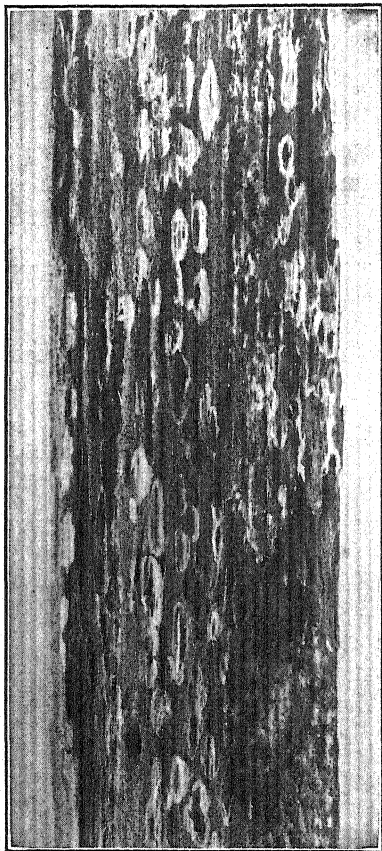


FIG. 143. — A piece of wood, split from the heart of a Douglas Fir tree, showing the typical delignifying rot caused by *Trametes pini*.

found on the oak and other deciduous trees but has been seen on conifers. Among the species of fungi which are confined to the Gymnosperm or the Angiosperm hosts as the case may be, not all can attack all kinds of trees within the group with equal facility. For example, the dry-rot fungus, *Polyporus amarus*, is confined to the Incense cedar, *Libocedrus decurrens*. On the other hand, the ring-scale fungus, *Trametes pini*, occurs on species of *Abies*, *Larix*, *Picea*, *Pinus*, *Pseudotsuga* and *Tsuga*.

**Symptoms and signs.** — The decay caused by these fungi is of various kinds and located in various parts of the tree. The affected wood differs in color and texture for the various species of fungi. There is also a rather definite localization of the rot in certain parts of the tree in case of some of the species of fungi. Some species attack the heartwood only, others invade only the sap wood. Some attack both heartwood and sap wood. Some confine their activities to the basal part of the tree, others to the top, and still others

may attack at any point throughout the length of the tree.

There are two general types of fungi based upon their enzymatic action upon wood. One type is known as the delignifying type and the other the carbonizing type. Delignified wood looks white and fibrous (Fig. 143). Carbonized wood is friable like charcoal and usually red or brown in color (Figs. 144, 145). In some cases the wood is delignified

in small pockets or patches scattered thickly through the wood with the intervening areas apparently sound. The pits or pockets are at first white, later becoming hollow as the fibers disintegrate entirely, giving somewhat of a honey-combed effect (Fig. 143). Certain species break down and disintegrate the entire heartwood of the tree, finally leaving a large cavity in the center of the tree. In case of the carbonizing rots the red or brown rotten wood usually becomes much cracked or checked, sometimes breaking up into more or less cubical pieces. Badly decayed logs finally crumble into a powdery mass.

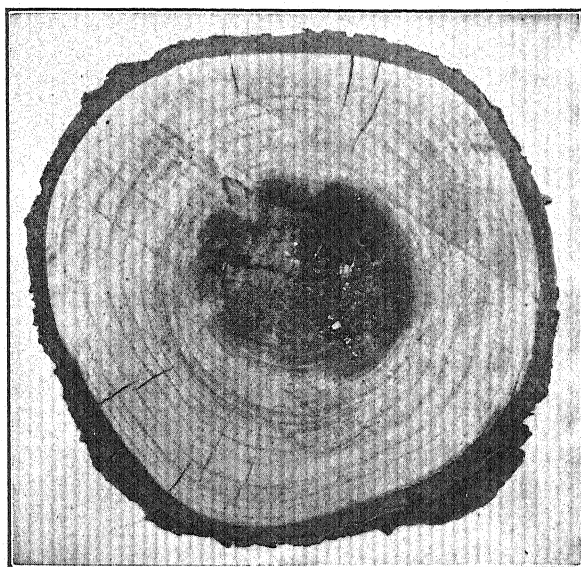


FIG. 144. — A brown, carbonizing rot caused by *Lenzites sepiaria* in a peach tree. (Photograph by Zeller, Ore. Agr. Exp. Sta.)

A striking sign of fungus attack consists of the bracket or shelf-like “conks” or sporophores which usually appear during favorable seasons on trees, logs or stumps that are badly decayed. These brackets vary with the species, in size, shape, color and other characteristics. The brackets are the chief means of identification for the different species of fungi, although some species produce a type of rot that is characteristic of the species and thus serves as a mark of identification in the absence of sporophores. Some species of wood-rotting fungi produce extensive white felts of mycelium in the cracks of the rotten wood. These felts sometimes become quite thick in the larger crevices, a thickness of 2 or 3 to 6 or 8 millimeters sometimes being attained. Large sheets of these

mycelial felts several feet in extent are occasionally found where large rotten logs are extensively cracked or checked, either along the line of the annual rings or of the medullary rays.

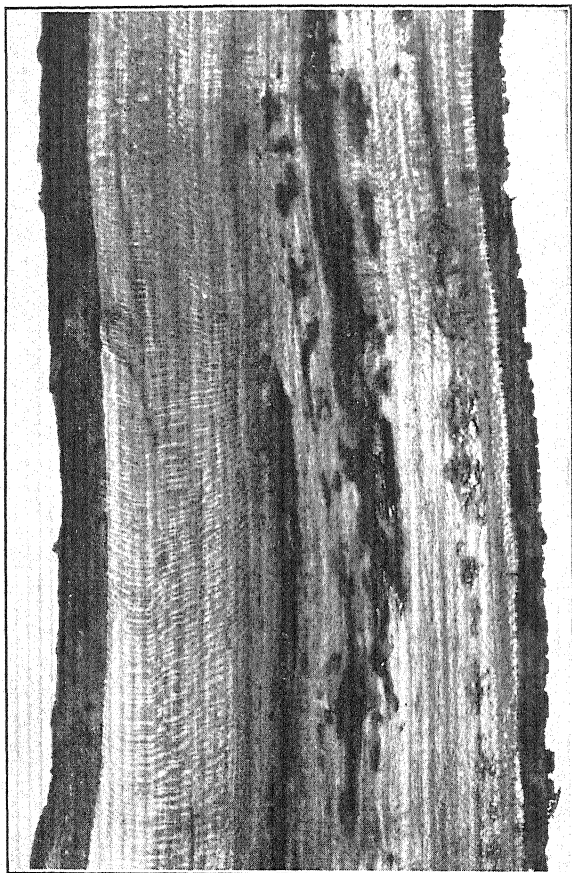


FIG. 145. — Brown-pocket rot caused by *Trametes subrosea* Weir. in a prune tree. (Photograph by Zeller, Ore. Agr. Exp. Sta.)

**Economic importance.** — The aggregate losses occasioned by the wood-rotting fungi are enormous. There are three different phases or types of damage that should be considered, namely, the damage done to standing timber in the forests, the injury caused by these fungi in orchards, and the toll taken from manufactured lumber products in decreased durability when exposed to the weather.

*In the forest.* — It is impossible to obtain exact figures on the amount

of loss caused by all the important wood-rotting fungi in the forest. In large lumbering operations it is possible, however, to get estimates based upon the amount of cull in logging and at the mill when the logs are sawed into lumber. In the Pacific Northwest the Douglas Fir, *Pseudotsuga taxifolia*, is the most important timber tree, occurring in enormous stands, much of which is overmature. Here the most serious decay is the pecky wood-rot caused by *Trametes pini* (Fig. 146). It is

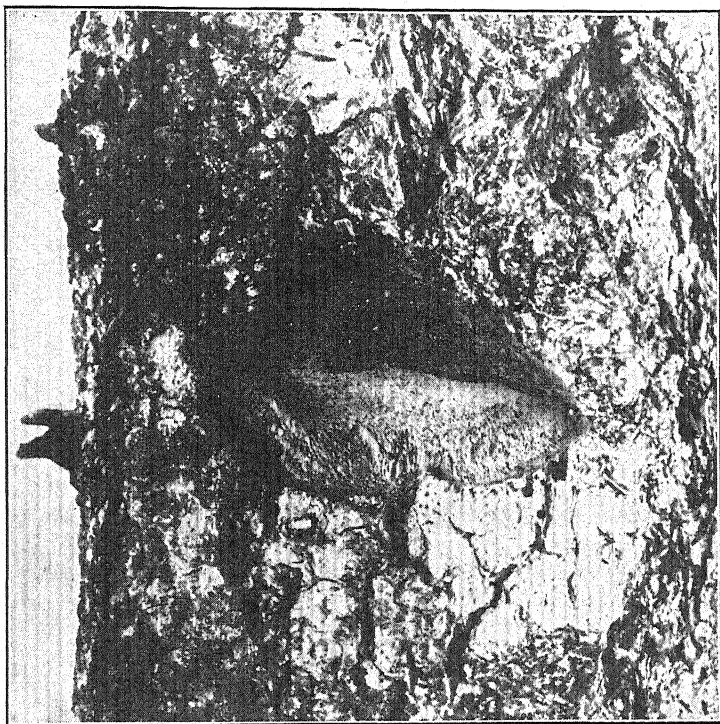


FIG. 146. — Sporophore of *Trametes pini*, a wood-rotting fungus, on a Douglas Fir tree, *Pseudotsuga taxifolia*.

stated (4) that in many of these stands of Douglas Fir there is a loss of 20 per cent in harvesting the lumber crop. In some cases the cull loss may reach 50 per cent or more. In such cases the loss in harvesting is so great that it becomes prohibitive since such stands cannot be logged at a profit. When one reflects that the stand of this species in the states of Washington and Oregon is estimated at 505 billion feet it becomes evident that this loss is enormous. It should be noted that wood decay in the forest is serious only in mature or over-mature stands.

In general, trees are quite free from decay until at least 75 to 100 years old and may escape infection until much older.

*On manufactured lumber and timbers.* — After a tree is made up into lumber it is safe from decay only if the products are kept under conditions unfavorable for the growth of wood-destroying fungi. Lumber or timbers exposed to the weather and especially in contact with moist

soil or other source of moisture are subject to decay. The loss from decay of wooden fence posts alone on the farms of the United States is enormous. Railroad companies lose millions of dollars annually because of the shortened life of ties due to decay. If telegraph and telephone poles did not rot off at the surface of the ground thus necessitating replacement every few years, the saving to these corporations would be tremendous. Where lumber companies stack lumber carelessly and leave it for a long period of time considerable loss may result through invasion of the stacks by fungi (10). A form of dry-rot (5) sometimes gets into the foundation timbers of buildings and serious damage results before it is discovered.



FIG. 147. — Prune tree with broken down limb due to heart-rot. (After Zeller, Ore. Agr. Exp. Sta. Crop Pest Rept., 1915-20.)

*In orchards.* — Fruit trees are susceptible to many of the same fungi which cause decay in the forest. The damage done in the orchard is of a different nature, of course, from that occurring in the forest. In the orchard a crop of fruit is the important thing and anything which injures the tree in such way as to decrease either the quantity or quality of the fruit is harmful. A fruit tree which is badly infested with heart-rot is more or less

devitalized and cannot produce as good a crop as a healthy tree. Some decay fungi also invade the sap wood thus killing branches or the whole tree. An important effect of wood decay is the weakening of the tree so that branches are more easily broken by wind or a heavy crop. It is not uncommon to see fruit trees with many or even all of the limbs broken down because of the weakening effect of

heart-rot (Fig. 147). A certain type of canker sometimes appears on trees that are badly devitalized by heart-rot fungi. Such cankers are caused by the fungus coming to the surface and killing the bark. They frequently occur around pruning cuts or other wounds (Fig. 148).

**Morphology of wood-rotting fungi.** — This discussion will apply in general to any of the four common families of the wood-rotting fungi belonging to the Basidiomycetes, namely, the Polyporaceae (Pore-fungi), Hydnaceae (Tooth-fungi), Agaricaceae (Gill-fungi), and Thelephoraceae (Fungi with a smooth spore-bearing surface). The vegetative mycelium of any of these forms is filamentous, much branched, septate, and bears the "clamp-connections" characteristic of many of the higher Basidiomycetes. The hyphae readily penetrate wood, growing largely in the cell cavities, especially in the tracheae and tracheids, but readily penetrating cell walls, thus being able to

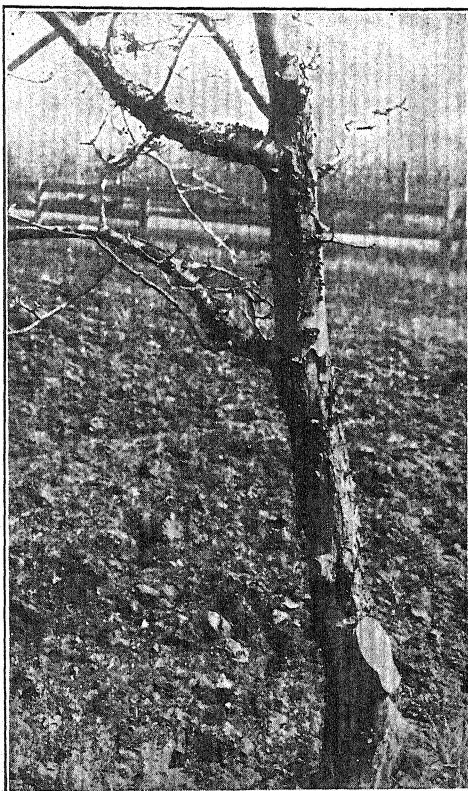


FIG. 148. — A shaggy-bark, cankered condition in an apple tree due to invasion by a wood-rotting fungus. (After Zeller, Ore. Agr. Exp. Sta. Cir. 73.)

spread transversely as well as longitudinally in the stem (Fig. 149). The sporophores or fruiting bodies of these fungi are masses of fungous tissue which appear on the surface of the host or substratum and acquire many different shapes and varying characteristics. The most common forms of sporophores are the brackets or shelf fungi (Figs. 146, 151, 152), and the mushroom or toadstool forms. In many cases, however, the fruiting body is a crust which spreads out in a flat layer, clinging closely to the sub-stratum without any projecting or shelving structure of any kind. Such fruiting bodies are said to be resupinate.



The function of the sporophores is to bear spores. It is in the spore-bearing surface that the distinguishing characters of the four families mentioned above lie. In all forms the spores are borne on the under surface. In the Polyporaceae this surface is covered with pores of varying size and shape depending upon the species. In the Hydnnaceae the under surface is covered with projecting teeth. The under surface of the sporophore in the Agaricaceae is thrown into numerous folds or



FIG. 149. — Longitudinal section of wood showing the mycelium of a wood-rotting fungus in the cell cavities and penetrating the cell walls.

gills as seen under the cap of the common mushroom. The Thelephoraceae are distinguished by the fact that the spore-bearing surface has neither pores, teeth, nor gills but is smooth. These four characteristics are reflected in the common names applied to the four families indicated above. In all four cases the spores are borne on the typical basidium of higher basidiomycetes (Fig. 150 B). In the pore forms the basidia are produced in a layer lining the inner surface of the tubes or pores and the spores upon being dislodged from the basidia fall from the pores and are then disseminated by various agencies. In the gill forms both sides of each gill are covered with basidia while in the tooth

fungi the basidia completely surround each tooth. In the smooth-surfaced fungi the entire under surface of the sporophore is covered with basidia.

**Life history.** — The length of time the mycelium vegetates in the host tissue before fruiting varies with the different species and with conditions. In some cases the fungus may be present and spreading throughout the heart-wood of a tree for many years before producing the first sporophore. In other cases only a comparatively short time, a few months or a few years, intervenes between the time of infection and the time at which the first fruiting bodies are produced. The spores may be disseminated by wind, insects, birds or rodents. On living trees infection takes place only in wounds where dead wood is exposed. On dead trees and manufactured lumber, of course, infection can occur anywhere, provided moisture and temperature conditions are favorable.

In the forest there are many agents that produce wounds through which infection may take place. One of the most important causes of wounds is fire, which frequently leaves open scars through which the wood-rotting fungi easily enter and reach the heart-wood. This is a common avenue of entry for those fungi which cause rots in the base of the tree, as, for example, the red root-, and butt-rot caused by the velvet-top fungus, *Polyporus schweinitzii*, in some of our coniferous trees. Another very important type of infection-court is wounds occasioned by the breaking-off of branches. Old branch stubs and knot-holes expose dead wood, particularly heart-wood, and afford ideal places for infection to occur. Broken branches in the forest are due to a number of causes,

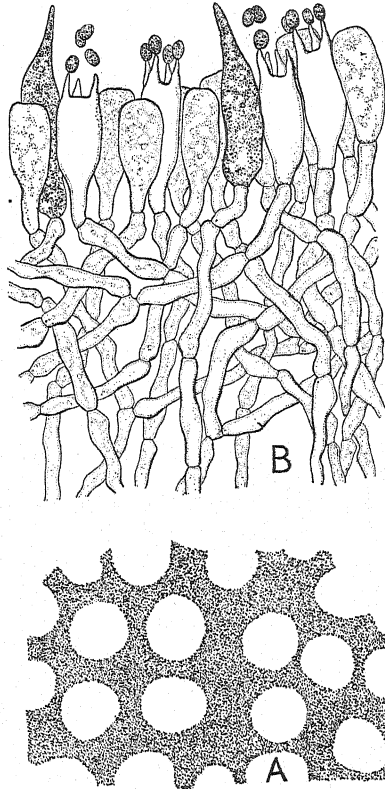


FIG. 150. — A, cross section of the pores of a bracket fungus (Polyporaceae). B, sectional view of a portion of the hymenial layer immediately surrounding one of the pores in A, showing basidia, basidiospores and two sterile cells, cystidia.



as wind, snow and ice. Lightning also causes wounds through which fungi may enter.

In orchard trees the wounds are largely man-made. In pruning operations in old orchards, quite large branches are sometimes cut off, thus leaving a large wound where infection may easily occur. Carelessness in other orchard operations may result in large areas of bark



FIG. 151. — Sporophore of *Fomes pinicola* on a peach tree. (Photograph by Zeller, Ore. Agr. Exp. Sta.)

being knocked off. If these exposed areas do not heal quickly, wood-rotting fungi may enter there. In addition to wounds due to man's activities, broken branches may result from storms or from an overload of fruit. Canker diseases such as European canker or anthracnose may expose areas of dead wood which become subject to invasion by wood-rotting fungi.

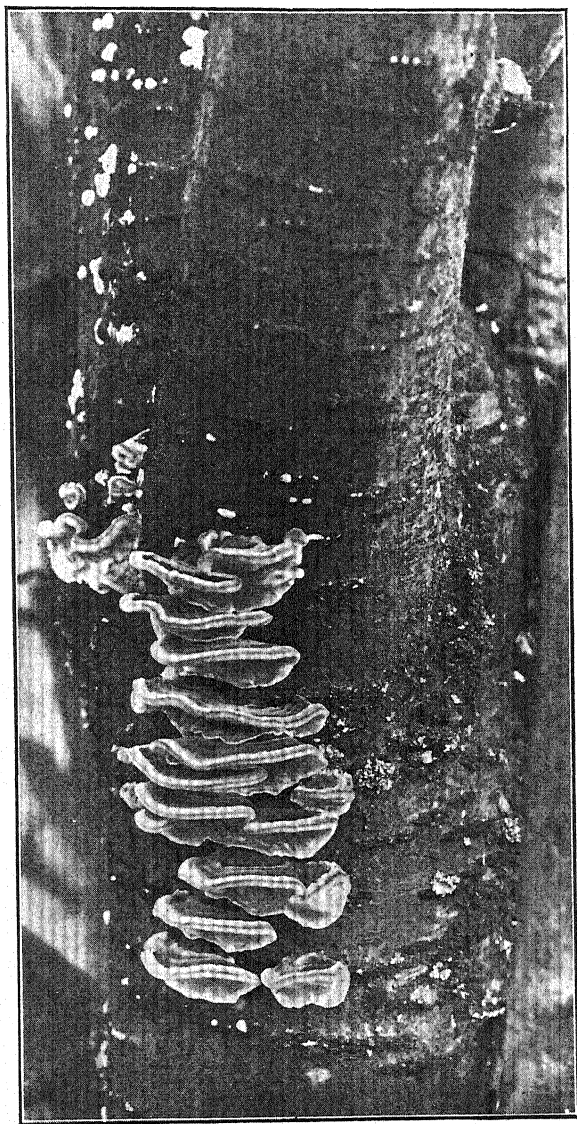


FIG. 152. — Sporophores of a common wood-rotting fungus, *Polystictus hirsutus*, on a cherry tree. (Photograph by Zeller, Ore. Agr. Exp. Sta.)

**Control.** — In view of the preceding discussion the question of control can best be taken up under three heads: (a) in the forest; (b) in the orchard; and (c) with reference to manufactured forest products.

In the virgin, overmature forest it is not possible to do anything towards control of wood-rot diseases. After the fungus has entered the tree it is manifestly impossible to cure the tree. It is, likewise, not feasible to prevent new infections from occurring, since wounds of various kinds, some of them beyond the power of man to prevent, are bound to occur, and since the fungi are already abundantly scattered throughout the forest. In places where the original forest no longer exists and where intensive forest management prevails, something can be done to lessen the losses from this source. (It has been found that for each species of forest tree there is probably an age limit up to which there is very little wood-rot infection and beyond which the percentage of rot rapidly increases.) When this age limit has been determined for any species, the cutting cycle in such stands should be fixed at such an age as will give the best yield of sound timber with the least loss from decay. Generally speaking the age limit for this cutting cycle will be determined by the age up to which the annual increment in board feet exceeds the annual loss from decay, and beyond which the loss equals or exceeds the increase (14). When such a system of forest management has been put into effect the loss of timber from wood-rots will be reduced to a minimum.) Such a system is already in operation in Europe and can be put into effect in the older sections of the United States where new crops of timber are being grown. Farmers with private wood lots also can practice such methods. In the large forest areas of the Pacific Northwest, of course, this stage has not yet been reached but in the future as the virgin forests disappear and reforestation occurs much can be done to cut down the wood-rot losses. At present the practice of leaving old diseased trees as seed trees in cut-over areas should be discouraged.

In the orchard the control problem resolves itself into two phases: (a) The prevention of wounds as far as possible, and (b) the treatment of inevitable wounds in such manner as to prevent infection. In so far as possible, fruit trees should be pruned when young in such manner as to make it unnecessary to prune out large branches when the tree grows older. The removal of such small twigs from time to time as may be necessary to keep the head of the tree properly thinned does not expose the tree to serious danger from wood-rots. The decay fungi do not enter readily through small pruning wounds, but the pruning out of larger branches exposes more heart-wood and offers a more accessible path to the heart of the tree. If it becomes necessary to prune out large branches the cut should be made flush with the trunk of the tree to facilitate

healing. Long stubs should never be left projecting from the tree. But if large wounds do occur inadvertently from any cause it becomes necessary to take precautions to prevent fungi from entering such wounds. Some sort of disinfectant or protective covering must be applied to the wound. There are two general types of wound dressings in use: (a) the air-tight covering and (b) the air-porous type. The principle involved in the former is to apply some sort of paint, asphalt or grafting wax that will form an air-tight covering over the wound and thus exclude all fungous spores from the susceptible wood. The second type involves the application of some substance that is toxic to fungous spores, depending upon the fungicidal value of the dressing to kill spores after they lodge upon the wounded surface rather than a covering which will exclude them from the wound entirely. Either type will prevent wood-rot infection provided it meets certain requirements. The air-tight dressing must be a substance that will cover the wound completely, will not crack nor melt and run off. The air-porous dressing must be a substance that will stay on the wound for a long time and retain its toxic properties so as to offer a continuous protection to the wounded tree. Both types must be non-injurious to the living tissues of the tree in order that they may not interfere with the proper healing of the wound. It is difficult to find an air-tight covering that is satisfactory. If it does not crack or melt under the varying weather conditions the sap is apt to exude under the covering and cause it to separate from the wood more or less and permit spores to enter between the covering and the wood where they germinate readily under the moist conditions. Many think that an air-porous antiseptic is better than the air-tight covering for these reasons.

One of the best antiseptic dressings that has been devised is bordeaux paste. This paste may be made by the following formula:

Copper sulfate,  $1\frac{1}{2}$  pounds in 1 gallon of water.

Quick lime, 3 pounds slaked in 1 gallon of water.

Mix in equal parts and apply in a similar manner as white wash.

A more durable paste can be made by stirring raw linseed-oil (30) into commercial bordeaux dust. The dust is placed in a pail and the linseed-oil is added slowly while stirring. Enough oil is added to bring to the consistency of paint or whitewash. It may be applied with a brush as paint is applied.

For the preservation of posts, telephone poles, railroad ties and all timbers which are exposed to the weather, especially in contact with the soil, or used in other places where they are exposed to considerable moisture, a number of different preservatives have been tried. Some

of these are coal-tar creosote, zinc chloride, tar, and crude oil. The creosote seems to give the best results under most conditions. It may be applied with a brush, by dipping in an open tank, or applied under pressure in a closed tank. The pressure method gives the best results but is the most expensive. It is used by railroad companies in treating railroad ties. On the farm the most practical method is to set the posts in an open tank of creosote which can be heated to 180° or 200° F. and keep them there for an hour or more. The preservative can be applied with a brush the most cheaply of all, but this method is the least effective of the three. (For treatment of farm timbers see reference 38.) (For effect of preservatives on wood-rotting fungi see reference 37.)

For the control of the dry-rot fungi which attack the foundation timbers of buildings and sometimes also the floors and walls, the remedy is more ventilation under the foundations of the building and care to see that no wood comes in contact with moist earth. Leaky steam pipes in basements sometimes supply the necessary moisture for the growth of these fungi. A curious fact concerning some of these dry-rot fungi, for example, *Poria incrassata*, is the production of long, thick fungous strands many feet in length. These strands serve to conduct moisture from the source of supply for long distances so that if the fungus starts growth in a timber in contact with the soil, the fungus can spread up the wall timbers into the upper stories. The long, rope-like fungous strands act as siphons or wicks to transport water from the source of supply to the part of the fungus higher up, thus enabling it to rot the perfectly dry wood of the upper stories of a house.

*Tree surgery.* — Thus far the discussion has centered upon prevention of wood-rots rather than upon cure. While it is true that the successful treatment of a tree after it has been invaded by heart-rot fungi is a difficult and expensive operation, nevertheless the life of such a tree can be greatly prolonged by proper treatment. Tree surgery is being practiced more and more extensively, especially in the parts of the country which have been settled longest and where trees, because of scarcity, age, or for sentimental reasons, are highly valued. All of our older cities have many large, beautiful, old trees in their parks and cemeteries and along their streets. It would take centuries to replace some of these trees. Many of them have been invaded by heart-rot fungi. In such cases it is often considered wise to spend large sums of money to save these trees. The procedure consists in cutting, chiseling or gouging out all decayed wood, shaping up the cavities properly for drainage, and then filling with cement (40, 41). Branches in danger of breaking off are braced so that they cannot break. If this work is properly done by skilled workmen the life of the tree may be prolonged for many years.

Tree surgery may also be profitably practiced in orchards under certain conditions. Sometimes a valuable fruit tree can be filled and braced and its productive life greatly prolonged. In case of fruit trees, however, the deciding question is whether or not such surgical treatment can be done at a profit.

#### LABORATORY STUDY OF WOOD-ROTS

**A. Symptoms.** — Examine blocks of wood cut from different kinds of trees and showing different types of rot. (See text for examples.) Which ones show the white, delignifying type and which the red or brown carbonizing type? Which kind is friable like charcoal? Note that some are heart-wood rots and others are sap-wood rots. Some types of rot show a pitted or honey-comb effect, others are stringy or fibrous, while still others rot the wood in a uniform manner. If opportunity offers, go out into the field and examine trees which show knot holes or other wounds or scars where wood-rotting fungi have entered. Is any rotted wood exposed? Can you find a hollow tree or log? What made it hollow? If a recently logged-off area is available many different types of wood-rots together with the fruiting bodies of the causal fungi can be observed. Describe the different types of rot observed and illustrate by drawings.

**B. The fungus.** — Examine sections of wood invaded by wood-rotting fungi and note the distribution of the vegetative mycelium throughout the tissue. Are the hyphae confined to the cell cavities, or do they penetrate cell walls? If the sections have been treated with stains which differentiate lignified and non-lignified tissues, can you see any evidence of the delignifying action of the fungus? In which type of rot should such action be evident? **Draw.**

1. *The sporophore.* — Examine specimens of the bracket or shelf-like fruiting bodies of some of the more important wood-rotting fungi (see text for illustrations). Learn the names of a few important species and associate the sporophore with the type of rot caused by each species. Note the hymenial layer of the fruiting body. Is it of the pore, tooth or gill type? To which family does it belong? Where are the spores borne? Cut a vertical section through the whole sporophore and note the texture of the tissue. Are there zones or layers visible? Is the sporophore annual or perennial? **Draw.**

Examine under the microscope a thin section cut across the pores. Can you find the basidia and basidiospores in the lining layer of a pore? **Draw.**

2. *Life history.* — Look up the life history and find out how these fungi are scattered, where infection occurs and especially the relation of wounds to the heart-rot type.

**C. Notes.** — Write complete notes on the wood-rots studied.

#### REVIEW QUESTIONS

1. What causes wood to decay?
2. Of what economic importance are wood-rotting fungi?
3. How does a fungus get into the heart-wood of a living tree? Can this be prevented practically in the forest? In the orchard?
4. How do the fungi which attack fruit trees differ from those which attack forest trees?
5. Are wood-rotting fungi invariably harmful? If not, what good do they do?

6. To what action on the part of the fungus is wood decay due?
7. Is manufactured lumber subject to decay? Under what conditions? How prevented?
8. Is the presence of mycelium in the wood always indicated by fruiting bodies on the surface?
9. Are wood rots considered a very serious problem in orchards? Describe two control measures.
10. In old countries where intensive reforestation is practiced, how is extensive loss from wood-rots prevented?

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### Armillaria Root-rot

Caused by *Armillaria mellea* (Vahl) Fr.

This disease, caused by one of the "gill" fungi mentioned with the wood-rotting forms in the preceding exercise, has been known under various names, among which are the oak-fungus rot, crown-rot, shoe-string fungus rot, mushroom root-rot, and *Armillaria* root-rot. The last-mentioned name is preferred by some to designate the root-rot caused by this specific mushroom since there are other closely related species of "gill" fungi which sometimes cause root-rots. In some regions the popular term "oak-fungus" is in common use because the strain of the fungus found in nature on oak trees is the form which commonly attacks fruit trees. The term "mushroom root-rot" has probably been used more widely than any other to designate this disease. This term, of course, is not specific and might be used to designate the root-rots due to any of the species of mushrooms which cause such troubles.

Root-rot caused by *Armillaria mellea* has been known in Europe for a long time and there is an extensive literature on the trouble in Germany and other European countries. The disease has probably been recognized in America since some time during the last quarter of the nineteenth century. In the United States it occurs extensively in the Pacific Coast states, the Southwest and in some of the central states.

**Hosts.** — The *Armillaria* root-rot fungus is capable of attacking a very wide range of plants including trees, shrubs and even plants of the herbaceous type. Among fruit trees known to be attacked are apple, plum, peach, cherry, orange, olive and English walnut. Additional plants on which the fungus has been found are grape, blackberry, raspberry, loganberry, gooseberry, currant, asparagus, strawberry and potato. In Europe many native forest trees are listed as hosts for *Armillaria mellea*, including the ash, alder, beech, birch, chestnut,

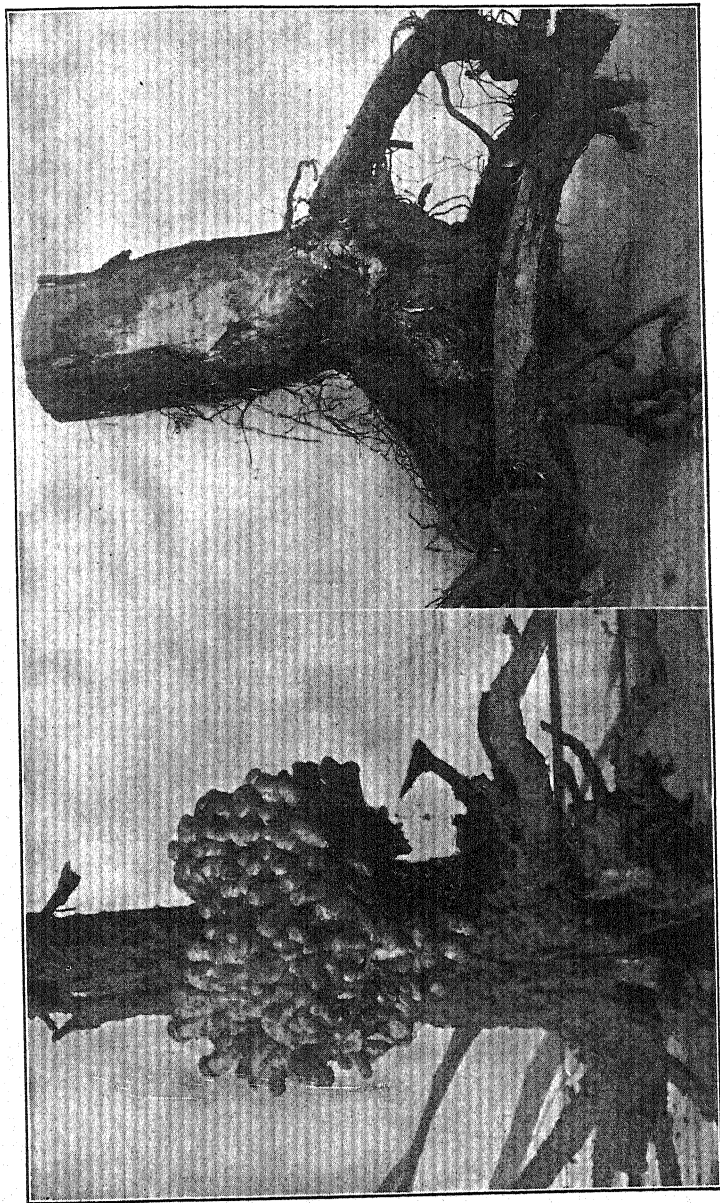


FIG. 153. — Left, young apple tree killed by *Armillaria* root-rot. The young (button) mushrooms are attached to the trunk at the ground line. Right, base of a prune tree killed by root-rot. Roots and crown are covered with rhizomorphic strands. Bark is stripped from the trunk and roots, showing white fan-like growth of fungus mycelium beneath. (After Barss, Ore. Agr. Exp. Sta. Crop Pest Rept., 1911-12.)

cotton-wood, oak, walnut, willow, fir, larch and pine. In the United States several forest trees, both coniferous and hardwood, have been listed as susceptible. Among these the oak seems to be most important since in most cases the fungus is reported to spread from the oak to the cultivated plants. The complete range of susceptible hosts is not known but there is evidence of striking resistance to *Armillaria* root-rot in certain trees. Horne (2) states that the pear seems to be immune. On the Pacific Coast there is a marked difference in resistance between the English walnut and the black walnut, the former being susceptible while the latter is very resistant.

**Economic importance.** — No figures are available on the total losses due to this disease, but in some localities it is undoubtedly considerable. The fungus works slowly but surely and while there are no spectacular outbreaks as with other types of diseases such as stem rust of wheat or late-blight of potato, yet in the course of a few years a considerable percentage of trees in an orchard may be killed. The fact that the disease spreads from tree to tree in the orchard, if allowed to go unchecked, threatens the complete destruction of an orchard in the course of time unless strenuous efforts are made to eradicate the disease. It usually happens that the trouble is not discovered until it is too late to save the infected trees. Second only to the damage caused in orchards is that occasioned in plantings of cane fruits such as blackberries and raspberries where the fungus readily spreads along the row and may kill a large number of plants in the course of a season. The most serious losses reported have been in apple orchards in Arkansas and Oklahoma, and on apples and prunes in the Pacific Coast states.

**Symptoms.** — The first outward indication of this disease is a yellowing or wilting of the leaves. When this happens the tree is usually already girdled and cannot be saved. Occasionally only one side of the tree may show this sickly appearance. This indicates that a root or roots on one side of the tree are rotted while other roots are still healthy. The real diagnostic symptoms, however, are found in the roots and crown of the tree, mostly below ground but extending for a few inches above the ground line. If the bark on the roots of a dying tree and around the base of the trunk is cut away white flakes of mycelial growth may be found throughout the dead bark and especially at the cambium line between bark and wood where extensive sheets of mycelial felts occur (Fig. 153). On the surface of the diseased roots there is usually an extensive development of shoe-string-like fungous strands known as rhizomorphs (Fig. 153). These "shoe-strings" are composed of compactly woven mycelial threads in much the same manner as a rope is composed of hemp fibers. They are white within but black on the

surface, much branched, and range from about 1 to 2 mm. in diameter. They are attached to the surface of the roots at many points and can be distinguished from the natural rootlets of the tree upon careful examination by noting the various characteristics mentioned above. The final sign of the disease is the appearance of clusters of the mushrooms at the base of the tree (Figs. 153, 154). These appear in the autumn on trees that are very sick or even dead from the disease.



FIG. 154. — Oak tree with cluster of the mushrooms of *Armillaria mellea* at base. (Photograph by Barss, Ore. Agr. Exp. Sta.)

**The fungus.** — The *Armillaria* root-rot is caused by *Armillaria mellea*, one of the Agaricaceae, or gill-fungi. Other species of this family are known to cause similar root-rots though *A. mellea* is apparently the most important of these root-rotting forms. A species of *Hypholoma* is sometimes found causing damage of this kind but this fungus is not accompanied by the rhizomorphic strands associated with

*Armillaria*. The life history of *Armillaria* is similar to the other gill-fungi in the production of the typical mushroom fruiting bodies with their numerous spores produced on the surface of the gills. In this case, however, the spores are not directly concerned in the infection of the living fruit trees or other cultivated plants. Apparently the rhizomorphs and the vegetative mycelium living in the dead roots of oak trees left in the soil of land recently cleared are the main source of in-

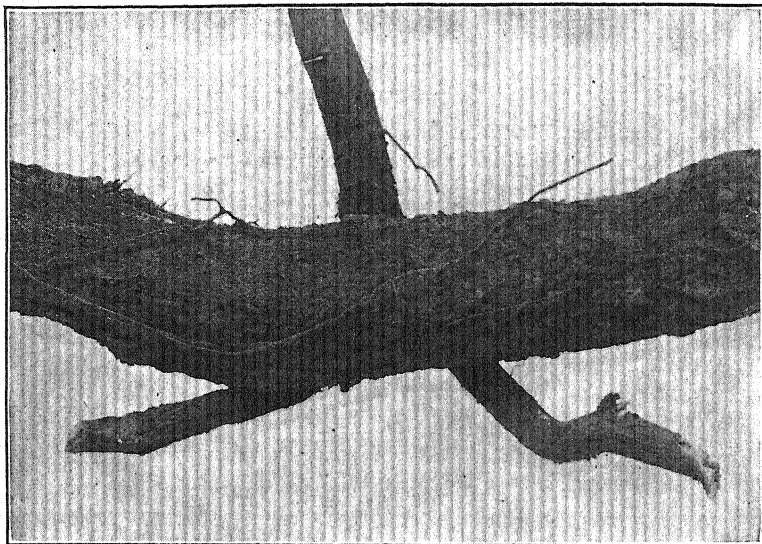


FIG. 155. — Apple tree root (forked) in contact with an old oak root infested with *Armillaria mellea*. The apple tree became infected at the point of contact. (After Zeller, *Phytopathology* 16 : 480. 1926.)

fection of the cultivated crops which are later planted on such cleared land. The exact manner in which the fungus penetrates the roots of healthy plants has caused considerable speculation. Some have suggested that a wound is necessary while others have supposed that wounds are perhaps not necessary. Horne (3) suggested that when a rhizomorph comes in contact with a root the hyphae of which the rhizomorph is composed penetrate the bark as individual hyphae. Zeller (7) has recently suggested that infection may occur in three different ways: through wounds; at points of contact of diseased roots with healthy roots (Fig. 155); and at the point of emergence of lateral roots where they rupture the bark.

**Control.** — The control of this disease involves the problem of prevention, since trees which are already badly diseased can rarely be saved

and then only at great expense and trouble. Since the fungus comes originally from the native host, the oak, the first precaution consists in avoiding the planting of susceptible crops, especially orchard trees, on land recently cleared of oak unless all stumps and roots are first removed from the land. The more rigidly this precaution is complied with the less danger there will be from root-rot later on. In case the disease is discovered in a planting the safest plan is to uproot and burn all in-

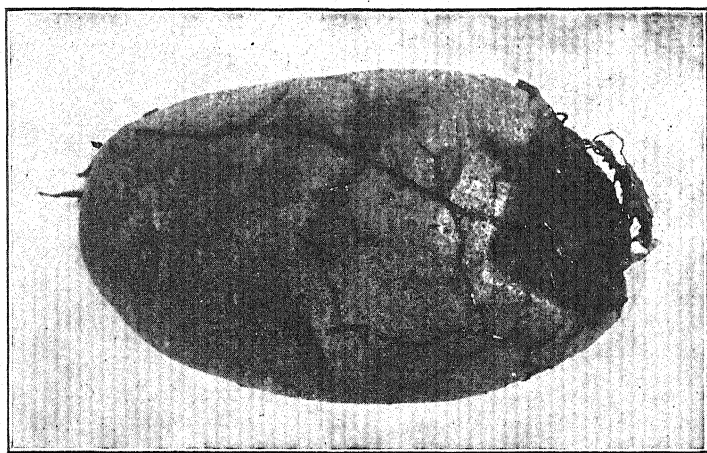


FIG. 156. — Potato tuber infested with the *Armillaria* root-rot disease, showing rhizomorphs attached. (Photograph by Bailey, Ore. Agr. Exp. Sta.)

festes trees. It is best not to plant susceptible trees in the same spot for a few years. Sometimes individual trees can be saved if discovered before all roots are infested or before girdling of the trunk occurs. Treatment consists in exposing the root system at the base of the tree and pruning off all dead roots and infested bark, after which a good wound antiseptic should be used and the roots left exposed to the sun for a time. Digging a trench two feet deep around a diseased tree at the outer limits of its root system has been suggested as a means of preventing spread from tree to tree through contact of root systems.

#### LABORATORY STUDY OF *ARMILLARIA* ROOT-ROT

**Symptoms.** — The stumps of young trees killed by this disease may be dug out and brought into the laboratory for careful study. Note the rhizomorphs attached to the surface of the roots. Peel sections of bark from the roots and base of the trunk. Observe the mycelial felts or fans spread out between the bark and the wood in the cambial zone. Also note that flakes of the mycelium can sometimes be seen scattered throughout the bark. Examine the wood and note the extent to which it is decayed. In advanced stages the roots readily break off. How far above the ground



line in the trunk does the fungus extend? If it is possible to observe diseased trees in the field during the growing season, note the symptoms exhibited there. Make drawings to illustrate all symptoms observed.

**The fungus.** — Mount bits both of the mycelial felts from the bark, and of the rhizomorphs. What is the microscopic structure of these vegetative parts? **Draw.** Examine the fruiting sporophores and compare with any typical mushroom. Is this sporophore a typical mushroom in form and structure? What are the distinguishing characters of the genus *Armillaria*? Examine sections of the gills under the microscope. **Draw.** Where are the spores borne? What part do the spores play in reproduction? What is the function of the rhizomorphs?

**Notes.** — Write notes embodying the results of your studies on this disease.

#### REVIEW QUESTIONS

1. What are the native hosts of *Armillaria mellea*? The cultivated hosts?
2. How is the fungus transmitted from tree to tree?
3. Describe the above-ground symptoms of the disease. The underground symptoms.
4. What is the most effective method of prevention in the orchard?
5. Is there any cure for this disease? If so under what circumstances and in what manner can it be applied?

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## CHAPTER XX

### DISEASES CAUSED BY FUNGI-IMPERFECTI

We have already noted that many fungi have an asexual or conidial spore form to which we have sometimes referred as an imperfect stage. The brown-rot of stone fruits, apple-scab, black-rot canker, and wheat-scab may be cited as examples of diseases caused by fungi which possess a conidial or imperfect stage in addition to the perfect or ascospore stage. We have also learned that certain of the Ascomycetes possess only one spore form, the ascospore. On the other hand we are now to learn that a great many fungi possess only a conidial or imperfect stage in their life history. It would perhaps be more accurate to say that only an imperfect stage is known. The perfect stage of many such fungi may exist but has never been discovered or demonstrated to be generically connected with the known conidial stage. Some fungi which were formerly thought to possess only an imperfect phase have more recently been shown to have a perfect form as well. It was as late as 1902, for example, before the apothecial fruiting body of the brown-rot fungus was found to be just one stage in the complete life cycle of this organism. The conidiospores had been known for a long time previous to that date and until the apothecia were discovered the brown-rot fungus was classified as *Monilia*. Likewise the imperfect phase of the apple-scab fungus was at one time classified as a *Fusicladium* and was not transferred to the genus *Venturia* until the perfect stage was found and demonstrated to be a link in the life cycle of the apple-scab fungus. There are still many hundreds of species of fungi known only in the imperfect stage. All of these have been classified in various orders, families, genera, and species under the class Fungi-Imperfecti. Thus this class has been made a sort of pigeon hole for the more or less temporary classification of all those fungi which are known only in the imperfect stage. From time to time certain species will be transferred from this to some other class, as perfect stages are found for them. However, there will probably always be a considerable number of fungi which will never be transferred because it may be that some of them do not really possess a perfect stage at all.

The Fungi-Imperfecti are divided into three orders on the basis of the type of fruiting body in which the spores are borne. In the first order, the Sphaeropsidales, the conidia are borne in a *pycnidium* which



opens by a pore or slit (Fig. 166). Pycnidia bear a general resemblance to the perithecium of the Ascomycetes except that the spores are borne on conidiophores within the pycnidium, instead of in asci. In the second order, the Melanconiales, the conidiospores are borne within a more or less saucer-shaped fruiting pustule known as an **acervulus** (Fig. 172). In the third order, the Moniliales, the spores are borne more superficially than in either of the preceding orders, sometimes in tufts, and in other cases scattered at random over the surface of the substratum (Fig. 175). Examples of these three types will be found in the succeeding exercises.

### Apple-blotch

Caused by *Phyllosticta solitaria* E. and E.

Within the last twenty-five years this disease has come into great prominence as a serious malady of the apple in a large section of the United States. Previous to 1902 it apparently was of minor importance. At least it had not attracted the attention of plant pathologists or fruit growers to any considerable extent before that time. During recent years records indicate that its range is spreading and the losses due to it are increasing. As the disease gradually came to be noticed by growers in different sections various names have been applied to it, including fruit-blotch, star-fungus, cancer, dry-rot, black-scab, late-scab, and apple-blotch. During recent years the name "apple-blotch" has been generally accepted and used as the correct popular name for the disease. This name is suggestive of the characteristic symptoms on the fruit and seems to be a fitting and acceptable common name by which to designate this trouble.

**History.** — In so far as known, America is the original home of this disease. The first record of its occurrence indicates that it was collected in Indiana (21) in 1893 on the leaves of the wild crab apple, *Pyrus coronaria* L. The causal fungus was named and briefly described by Ellis (4) in 1895. The wild crab is probably the native host of the fungus and no doubt it later spread to the cultivated apples. Clinton (2) first described the disease on cultivated varieties of apples in 1902. He found it quite prevalent in the orchards of southern Illinois at that time. Faurot (5) reported the presence of the disease in Missouri apple orchards in 1903. In 1907 Sheldon (20) stated that the fungus causing the blotch disease on cultivated apples is identical with the species collected on the wild crab apple several years previously, and which Ellis had named *Phyllosticta solitaria*. He did not prove this, however,

by artificial cultures. During the same year Scott and Rorer (17) demonstrated by isolation and inoculation work that the symptoms on all parts of the plant, fruit, leaf, and stem are caused by one and the same fungus. Previous to this time there had been no definite proof that the leaf-spot and the fruit-blotch, for example, are simply two phases of the same disease and not two separate and distinct diseases. The first experimental work on control was done in Illinois in 1903, where Crandall (3) tried out the relative effectiveness of bordeaux dust and liquid bordeaux for the control of apple diseases including blotch. In 1906 Scott and Quaintance, in Arkansas, conducted a set of spraying experiments especially for the control of apple-blotch. About 1912, considerable interest was manifest in the possibility of controlling blotch by dormant sprays. Since 1909, however, the chief interest in connection with control has centered in the relative merits of lime-sulfur and bordeaux. In 1917, ten years after the inoculation work of Scott and Rorer, Roberts (12) repeated the culture experiments and confirmed the findings of these two men. In Indiana, in 1922, Gardner (6) found that the majority of apple-blotch cankers on twigs result from invasion by the fungus, already present in petioles and bud-scales, which grows across the abscission layer before leaf-fall so that mycelium of the fungus is left in the tissue of the leaf scar when the leaf falls.

**Geographical distribution.** — In 1902 the disease on commercial apples was known in Illinois, Missouri and Arkansas. In 1907 it was reported from North Carolina. In 1908 Kentucky and Oklahoma were added to the list of states in which blotch had been found in orchards, and observations made the same year indicated the general distribution of the disease throughout the region extending from Arkansas and Missouri on the west to North Carolina and Maryland on the east. In 1910 it was reported from Southern Indiana and Ohio, in 1912 from New Jersey, and in 1924 from New York. At the present time it is pretty generally distributed throughout the eastern half of the United States, particularly the central and southern part of this region. The approximate boundary of this infected region extends from New Jersey and Georgia to Nebraska and Texas. Blotch is not confined absolutely within this territory but is not serious outside of it. The northern boundary of serious infection is approximately along the 40th parallel which runs just north of Columbus, Ohio; Indianapolis, Indiana; and Springfield, Illinois. Scattered infections, however, are reported as far north as the northern boundaries of these states. The zone of greatest prevalence runs through Kansas, Missouri, Arkansas and Tennessee. It is very severe in a belt along the Ohio River also, and reports of severe infections have come from as far south as Mississippi.

**Hosts and relative susceptibility.** — This disease is confined almost exclusively if not entirely to species of the genus *Malus*. Several species of wild crab apples are susceptible. The blotch fungus readily attacks a great many of the cultivated varieties of apples. There is a great variation in susceptibility of the different varieties of apples. An important point to note is the fact that any one variety is not equally susceptible to both the fruit-blotch and the canker phases of the disease. Guba (9) tabulates a long list of apple varieties with respect to their degree of susceptibility or resistance to both the fruit-blotch and the bark-canker phases of the disease. His classification of these varieties follows:

### I. Susceptibility to fruit-blotch

1. *Very susceptible.* — Arkansas Black, Arkansas Red, Ben Davis, Benoni, Bentley Sweet, Chenango, Clayton, Domine, Duchess, Early Harvest, Ewalt, Fameuse, Gano, Gilpin, Harvest Pippin, Hawthornden, Huntsman Favorite, Krauser, Lansingburg, Lawver, Limbertwig, Maiden Blush, Mann, Missouri Pippin, Northwestern Greening, Oliver (Senator), Paradise Sweet, Red Astrachan, Rhode Island Greening, Rome Beauty, Royal Pearmain, Schockley, Smith Cider, Sops of Wine, Stark, Tolman Sweet, Wagener, White Winter Pearmain, Yellow Transparent.

2. *Moderately susceptible.* — Aiken Red, Baldwin, Bradford, Champion, Fink, Golden Russet, Ingram, Mammoth Black Twig, May of Myers, McAfee, McIntosh, Minkler, Northern Spy, Ralls Genett, Rambo, Roman Stem, Salome, Shannon, Willow Twig, Yellow Bellflower, Yellow Newton.

3. *Resistant or slightly susceptible.* — Delicious, Grimes Golden, Jonathan, Red June, Stayman Winesap, Wealthy, York Imperial, Winesap.

### II. Susceptibility to bark canker

1. *Very susceptible.* — Benoni, Bentley Sweet, Chenango, Duchess, Fameuse, Lawver, Mann, Missouri Pippin, Northwestern Greening, Smith Cider.

2. *Moderately susceptible.* — Baldwin, Ben Davis, Gano, Limbertwig, Maiden Blush, McIntosh, Oliver (Senator), Red Astrachan, Rhode Island Greening, Rome Beauty, Stark, Yellow Transparent.

3. *Resistant or slightly susceptible.* — Aiken Red, Champion, Delicious, Early Harvest, Fallawater, Fink, Grimes Golden, Huntsman, Ingram, Jonathan, Mammoth Black Twig, May of Myers, Minkler, Northern

Spy, Ralls Genett, Rambo, Red June, Sops of Wine, Stayman Winesap, Wealthy, Willow Twig, Winesap, Yellow Newton, York Imperial.

4. *Susceptible but degree uncertain.* — Arkansas Black, Arkansas Red, Bradford, Clayton, Domine, Gilpin, Golden Russet, Harvest Pippin, Hawthornden, Lansingburg, McAfee, Roman Stem, Royal Pearmain, Salome, Schockley, Shannon, Tolman Sweet, Wagener, White Winter Pearmain.

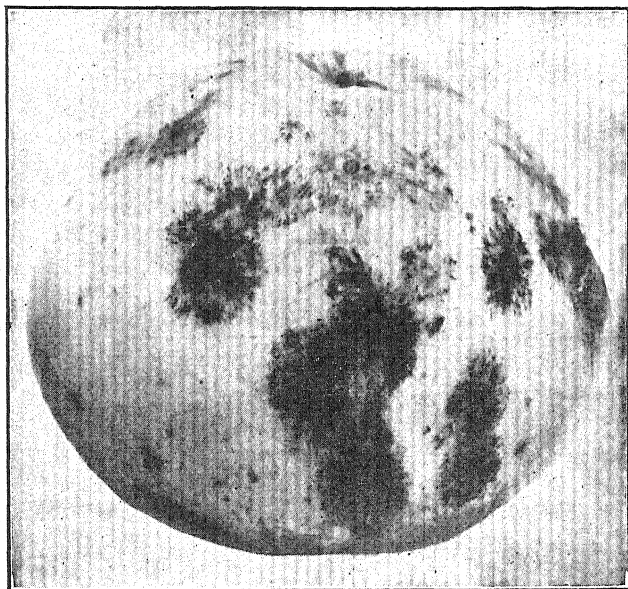


FIG. 157. — Apple fruit affected with blotch, showing the characteristic "star-fungus" effect. (Photograph by M. W. Gardner, Purdue Agr. Exp. Sta.)

**Symptoms.** — The attacks of this fungus result in characteristic lesions on fruit, leaves and twigs.

*On the fruit.* — The symptoms on the fruits are various but there are two main types. One of the most common and strikingly characteristic symptoms gives rise to the term "star fungus" which is sometimes applied to this disease. This type begins with the appearance of brownish fibers beneath the skin of the apple. These increase in number, become more dense, and at length there appear slightly elevated brownish blotches of varying shapes and sizes. Frequently these blotches are arranged in characteristic patterns with one blotch in the center and others distributed in a radiating manner around this center thus suggesting more or less vaguely, or definitely, a star with a variable

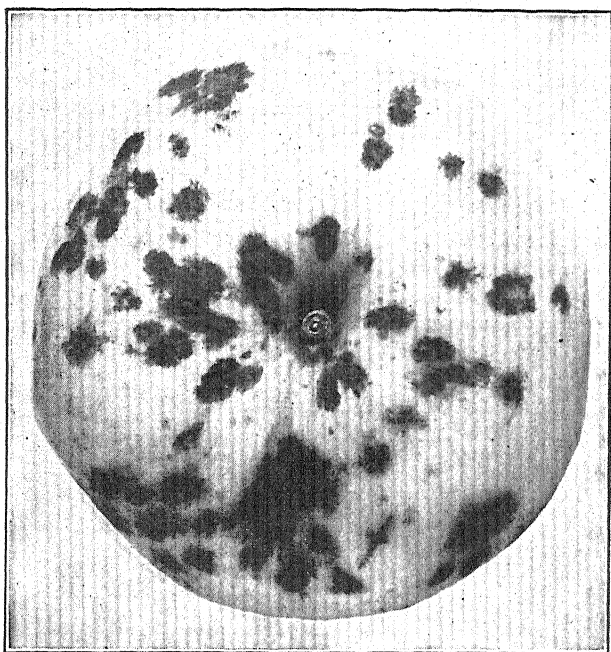


FIG. 158. — Small blotch lesions about the stem end of the fruit, probably resulting from rather late infection. (After M. W. Gardner, Purdue Agr. Exp. Sta. Bul. 267.)

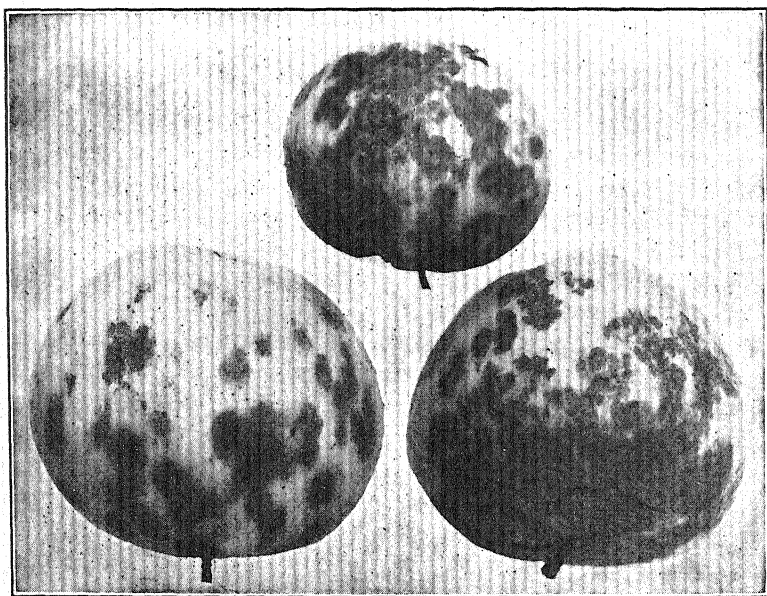


FIG. 159. — Apple-blotch on fruit, showing the cracking produced in advanced stages. (After M. W. Gardner, Purdue Agr. Exp. Sta. Bul. 267.) (458)

number of rays, usually more than five (Fig. 157). These rays may sometimes be suggestive of a feathery fringe of lighter color around a darker center. Sometimes the blotches begin as a dark, slightly sunken spot with or without a stellate fringe. The sunken portion increases in size until quite large blackened areas appear. Pycnidia usually are produced on these dark, sunken areas. In either type of lesion the fungus does not penetrate very deeply into the apple tissue. On severe lesions which start before the fruit is mature, deep cracks sometimes are formed in the same manner as such cracks appear in bad cases of apple or pear-scab (Fig. 159). Secondary rots sometimes enter at these cracks. On yellow apples the spots may be bordered with red. Raised, blister-like spots are produced on the Maiden Blush variety. Light colored fruits usually show the blotch lesions most conspicuously, while the lesions on dark colored varieties are less conspicuous.

*On leaves.* — On the leaf blade the typical lesion is a very small white spot, less than 1 mm. in diameter with usually one minute pycnidium in the center of each spot. This pycnidium is just large enough to be seen with the naked eye as a minute black point against the light background of the leaf-spot (Fig. 160). Lesions frequently occur on the mid-rib and petiole of the leaf as well as on the blade. (Figs. 161, 162.) Such lesions are much larger than the spots on the blade. They are usually elliptical in shape and 4 or 5 mm. in length or even longer. Pycnidia may also appear on the petiole or mid-rib lesions. In this case there may be several pycnidia per lesion instead of just one (Fig. 162).

*On the bark.* — Cankers appear on the twigs and spurs in late summer. The cankers may appear either at the nodes (leaf scars) or on the internodes (between leaf scars). On the current year's growth the young cankers first appear as dark, purplish, or black, raised or blister-like spots. As the twigs become older the spots become lighter in color and ultimately take on a tan color the following year (Fig. 163). In another year the canker becomes roughened and the dead parts begin to slough off. Cankers may increase in size for several years by reason of the marginal extension of the mycelium into healthy bark each succeeding year. Many small cankers may thus coalesce finally producing large roughened areas on the twigs and small branches (Fig. 164). At the end of the first season pycnosclerotia appear as black points protruding through the epidermis on the surface of the canker. When the canker begins to increase in size the next spring, due to resumption of growth by the mycelium, true pycnidia appear on the new extension zone around the margin of the old canker.

*Pathologic histology.* — Sections made through the cankered bark, when examined under the microscope, show the formation of successive



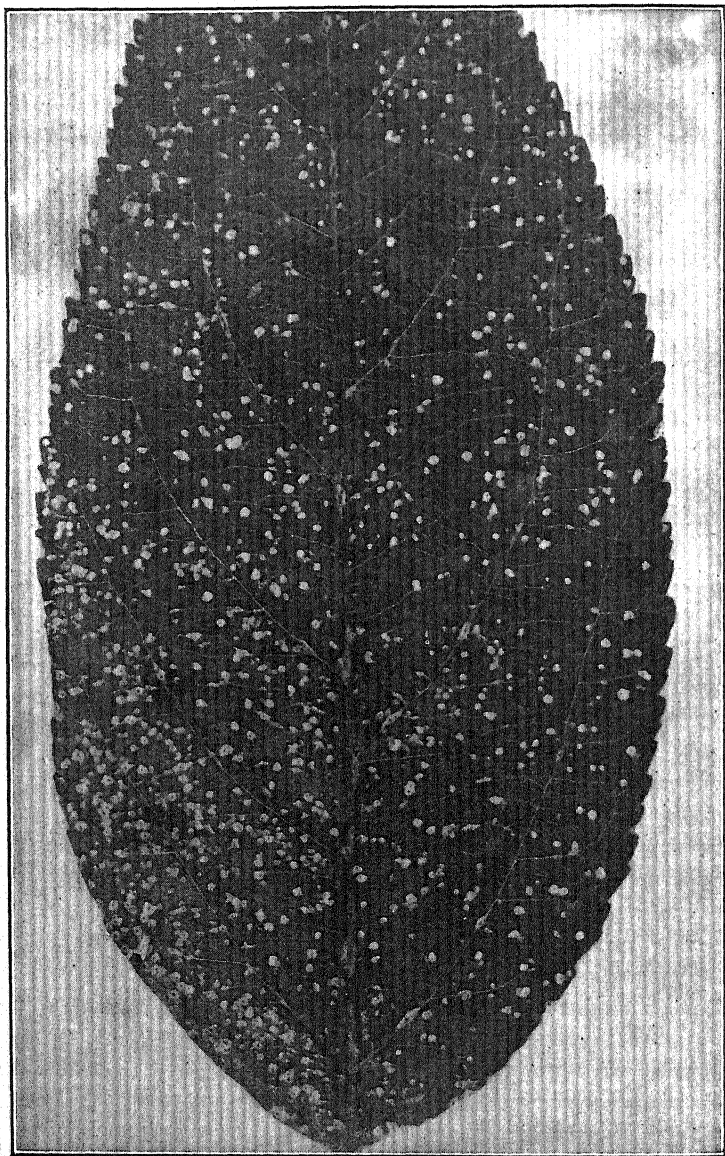


FIG. 160. — Apple-blotch lesions on leaf, magnified about two diameters, showing pycnidia.

zones of wound cork in the cortical region. These bands of cork effectively cut off the growth of the mycelium and prevent its penetration to the cambium in a majority of cases. These layers of cork are similar to the abscission layer which cuts off the leaf from the twig in the autumn and thus causes leaf-fall. In the blotch cankers it acts similarly for eventually the layers of outer bark thus cut off come loose and fall

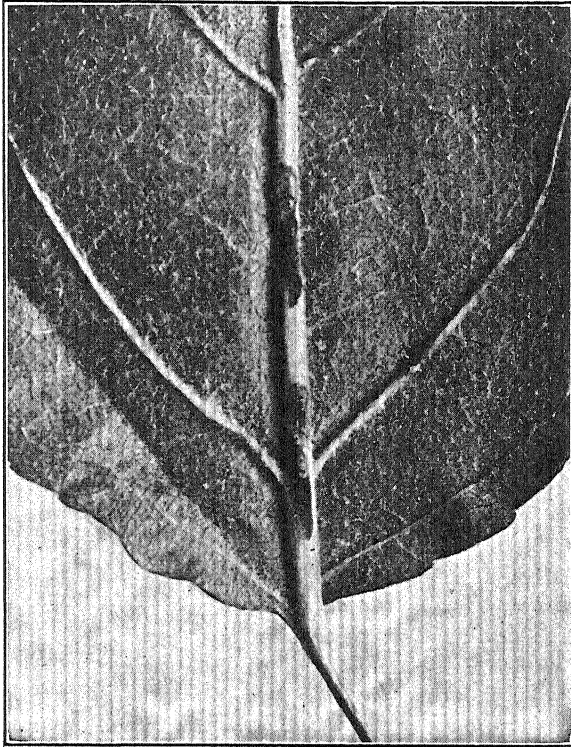


FIG. 161. — Apple-blotch lesions on the under side of the leaf mid-rib. (After M. W. Gardner, Purdue Agr. Exp. Sta. Bul. 267.)

away from the branch. Figure 165 shows these cork layers and the beginning of a crack at the margin of the canker which will ultimately extend along the entire length of the abscission layer, thus causing the dead area to fall away.

**Economic importance.** — The damage done by this fungus is of three types: (a) the fruit blemishes, (b) the twig cankers, and (c) the leaf-spots. The fruit-blotch is by far the most important type of injury. Severely blotched fruit, if marketable at all, usually brings a much reduced price. The canker form is not nearly as harmful to the tree



as many other types of cankers for the reason that ordinarily the mycelium does not penetrate to the cambium and thus open cankers with exposed dead wood are not commonly formed. Occasionally, however, the latter condition occurs and then open cankers or girdled twigs are produced. Nevertheless the extensive cankering of branches as shown

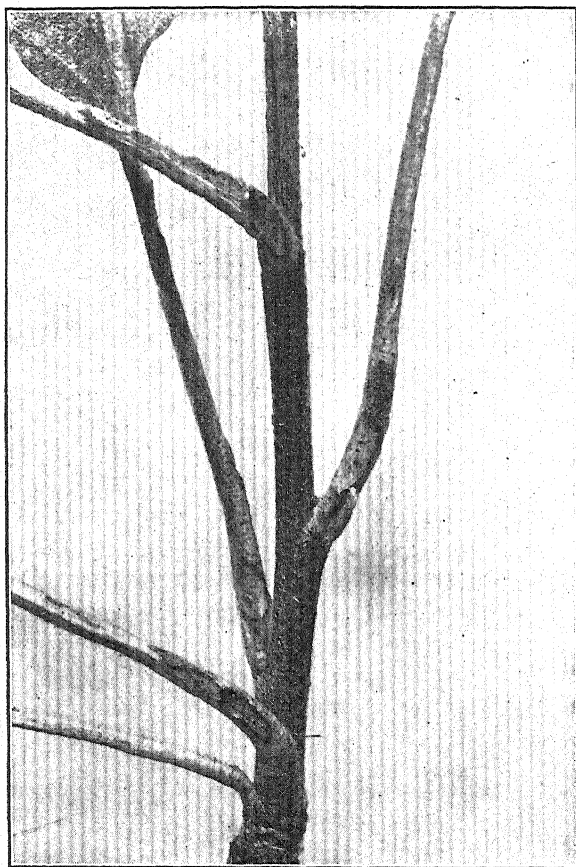


FIG. 162.— Apple-blotch lesions on leaf petioles. (After M. W. Gardner, Purdue Agr. Exp. Sta. Bul. 267.)

in Fig. 164 cannot help but injure the tree to a certain extent, even if no open cankers or girdled twigs are produced. The leaf-spot phase of the disease ordinarily does not cause serious damage but when it becomes severe enough to cause defoliation then of course more or less damage inevitably occurs.

The Plant Disease Survey of the U. S. Department of Agriculture (22)

is authority for the following estimates of losses in this country due to apple-blotch. In 1921 the loss in North Carolina was 10 per cent or 89,000 bu.; Illinois, 7 per cent or 226,000 bu.; Tennessee, 5 per cent or 45,000 bu.; and in Oklahoma, 10 per cent or 58,000 bu. The total loss for the United States in that year was .6 per cent or 668,000 bu.

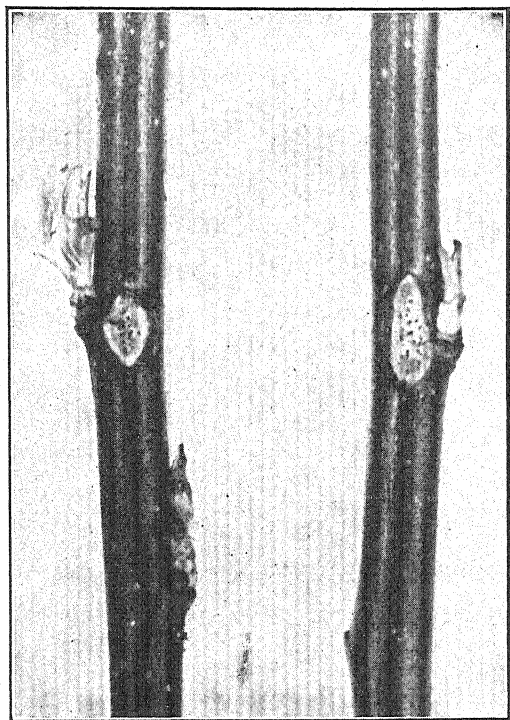


FIG. 163. — Apple-blotch on stems. Leaf scar cankers probably originating from infected leaf petioles. (Photograph by M. W. Gardner, Purdue Agr. Exp. Sta.)

In 1922 the loss in Pennsylvania was 1.5 per cent or 269,000 bu.; Virginia 2 per cent or 265,000 bu.; Ohio 5 per cent or 483,000 bu.; Indiana 2 per cent or 93,000 bu.; Illinois 6 per cent or 725,000 bu.; Kansas 10 per cent or 418,000 bu.; Kentucky 10 per cent or 874,000 bu.; Tennessee 8 per cent or 576,000 bu. The total for the entire United States in 1922 was 4,142,000 bu. In 1923 the loss for the entire country was 2,963,000 bu., and in 1924 it was 3,332,000 bu.

**Morphology of the fungus.** — The vegetative mycelium is much branched and septate. It penetrates the bark tissue extensively, until stopped by the formation of wound cork. It then forms dense masses

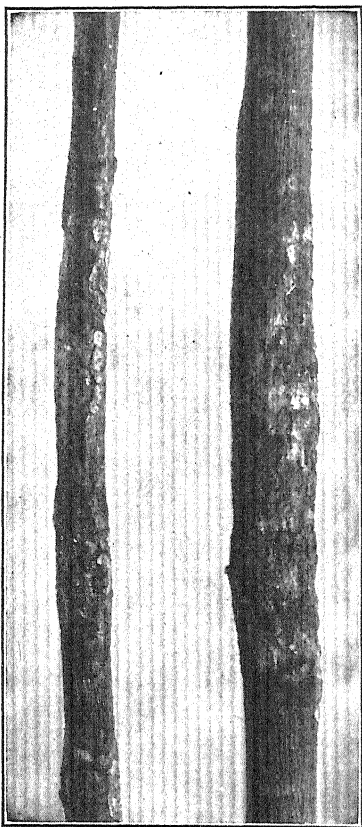


FIG. 164. — Old apple-blotch cankers on small branches.

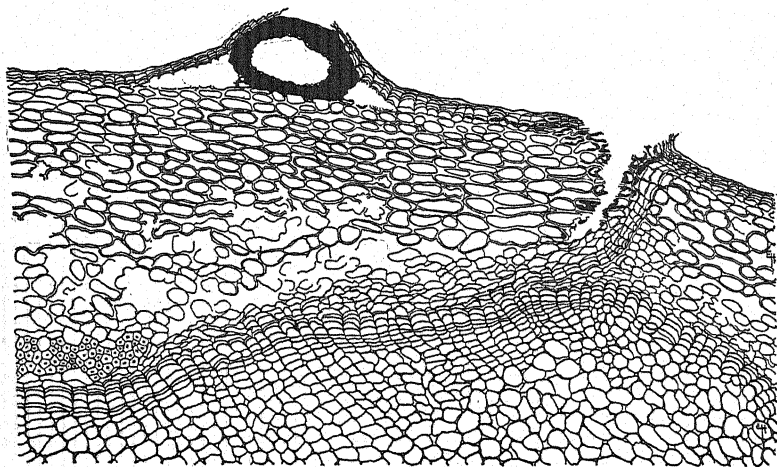


FIG. 165. — Section through bark of apple-blotch canker, showing wound-cork formation which acts as an "abscission layer" in cutting out areas of bark which die and slough off as shown in Fig. 164.

at intervals just beneath the epidermal layers. These masses of hyphae finally develop into pycnidia. There are several variations in the shape, size, and thickness of the wall of the pycnidia (9). Toward the end of the first growing season, bodies are formed which are known as pycnosclerotia. They are dense masses of mycelium which to all intents and purposes are sclerotia. They rest during the winter and the next spring develop into spore-bearing pycnidia (Fig. 166). Only the central part of the sclerotium becomes sporogenous leaving a very thick wall

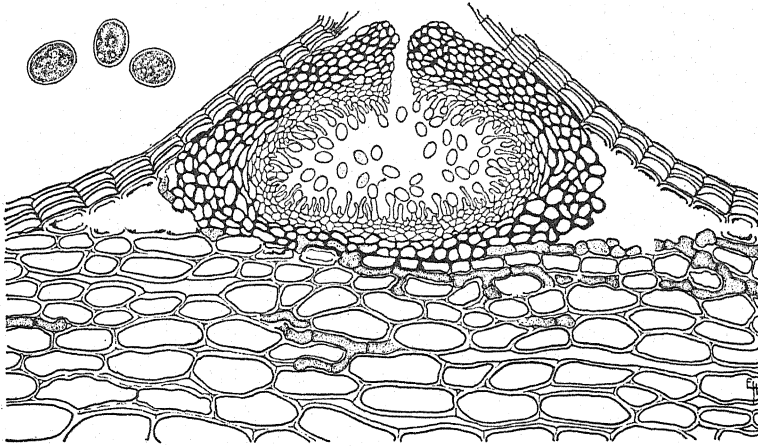


FIG. 166. — Section of pycnidium of *Phyllosticta solitaria*, from bark canker. At upper left, conidiospores enlarged.

surrounding the spore cavity. Early the next spring true pycnidia are produced on the new canker tissue which is formed around the margin of the old canker. These pycnidia have moderately thick walls, but not so thick as the walls of the pycnidia formed from pycnosclerotia. On the fruit the pycnidia are smaller and much flattened or elliptical in shape with still thinner walls. On the leaves the pycnidia are very small, nearly spherical in shape and have thinner walls than any of the other types. The smallest pycnidia range from 60 to 95  $\mu$  in diameter while the largest ones formed from pycnosclerotia measure 155 to 274  $\mu$  wide by 107 to 238  $\mu$  deep.

The cavity of the pycnidium is lined with conidiophores of varying shape and size which are usually unicellular, sometimes filiform, or short and broad. The conidia, or pycnospores, are unicellular, hyaline, guttulate, ovoid or broadly elliptic,  $7-11 \times 6-8.5 \mu$  (Fig. 166). No ascogenous stage is known although it seems likely that one exists.

**Life cycle.** — The chief means of hibernation is by the pycnosclerotia which overwinter in a resting condition and produce conidia in the spring. The fungus is said to remain alive for seven or eight years in the bark. Overwintering of pycnosclerotia and pycnidia may occur on fallen leaves and fruits but the chief place of hibernation is in the cankers. In the orchard the chief agency for disseminating spores from the twig cankers seems to be rain. Wind apparently plays a minor part in disseminating the blotch spores. The chief source of introduction into young orchards is on diseased nursery stock. Infection occurs directly from pycnospores on leaves, fruits and the internodes of young twigs. The majority of infections on twigs occur at leaf scars or at the bases of buds. Such cankers are the result of the invasion by mycelium which spreads downward from lesions near the base of the petiole and penetrates the abscission layer establishing itself in the leaf scar tissue before the leaf falls (6). Possibly some twig infection may be due to similar invasion from bud scales which become infected directly, as leaves do.

The first infections may occur shortly after blossoming time. Few infections occur earlier than two weeks following petal-fall. Most infections occur within a period of about seven weeks after the petals have fallen. Heavy precipitation is necessary to swell the pycnidia and aid in the discharge of the spores. A temperature of 77° to 86° F. is best for the germination of the spores and the development of the fungus, although spores will ultimately germinate at a lower temperature. Infections may, therefore, occur during long, cool, wet spells or during shorter periods of wet weather if the temperature is higher.

**Control.** — The nature of this disease and the life cycle of the causal fungus make it necessary to consider three chief items in discussing control measures, namely, (a) protective sprays, (b) pruning out diseased twigs, and (c) the use of disease-free nursery stock.

Since primary infections occur during the growing season the susceptible parts must be protected during the period of danger. Several sprays and dusts have been tried but the most satisfactory thus far used is bordeaux mixture (4-6-50 or 2-4-50). From 3 to 6 applications are recommended, beginning two weeks or less after petal-fall and repeated at two-week intervals. Lime-sulfur 1 to 40 may be used in the earlier sprays while the weather is cool to prevent the russetting which bordeaux is apt to cause, but lime-sulfur is not as effective for blotch control as bordeaux.

Since the chief means of hibernation is in the bark cankers it follows that if all of these were pruned out there would be much less chance for infection to occur on the new growth. Since most of the cankers are

on small twigs and spurs these can be pruned out without harming the tree. The outer bark of the rough cankers on larger twigs or small branches may be shaved off without disturbing the cambium if care is used.

Thirdly, the disease is readily transmitted on nursery stock. A few small cankers on nursery trees may easily escape detection when the trees are transplanted. If allowed to grow undisturbed these few diseased trees may serve ultimately to infect the whole orchard. Nurserymen should not use infected seedlings and should secure scion or bud wood from trees known to be free from blotch. Orchardists should not accept diseased stock for planting.

Speaking generally, for commercial orchards, a well planned and executed spray program is the surest and most practical method of holding this disease in check or even ultimately eradicating it from the orchard.

#### LABORATORY STUDY OF APPLE-BLOTCH

**A. Symptoms.** — Observe the symptoms of blotch on all parts of the plant.

1. *On twigs.* — Note especially the size of branches attacked. Does this appear to be a disease primarily attacking the smaller branches and twigs, or the larger branches and trunk? How deep into the cortex does the fungus penetrate? Does it kill to the cambium? Are open cankers with exposed wood produced by this fungus? Compare blotch cankers with other apple cankers studied. Find the fruiting bodies of the fungus on the cankered areas. What type of fruiting body does this fungus have? Make **drawings** to illustrate typical twig cankers.

2. *On fruits.* — Observe the typical blotch symptoms on fruits. Compare with apple scab. What are the earliest symptoms of blotch on fruits? Note the pattern presented by the brown markings. How deep into the skin do these young blotches extend? What are the characteristics of the older blotches? Are any fruiting bodies of the fungus present on the fruits? **Draw** to illustrate symptoms on fruit.

3. *On leaves.* — Note the size, color and distribution of the leaf-spots caused by the blotch fungus. Compare with the black-rot or frog-eye leaf-spot. Are pycnidia present? If so, what is their distribution within the spot? Are there any lesions on the petioles or mid-ribs of the leaf? **Draw** an entire leaf to show all symptoms and signs observed on any part of it.

**B. The fungus.** — How many spore stages are known for this fungus? Examine sections of diseased bark or leaf showing a vertical section of a pycnidium. **Draw.** Look up the life cycle of the fungus. How does it overwinter? When does infection take place? What is the source of the inoculum for the new infections? Compare the life cycle with that of the apple scab fungus.

#### REVIEW QUESTIONS

1. What is the geographic distribution of this disease in the United States?
2. Describe the life cycle of the causal fungus in detail.
3. What is the chief source of inoculum for the primary infections in the spring?
4. What is cork cambium? Where does it occur under normal conditions?
5. What is the abscission layer in leaves? How does it function?

6. What is the relation of the wound cork found in sections of blotch cankers to cork cambium and abscission layer? (See reference 8.)
7. At what points on twigs do a majority of the cankers occur? (See reference 6.) How is this fact accounted for?
8. What are the three chief methods of controlling this disease?
9. Why are not dormant sprays effective?
10. Why is it possible to cut out cankers on larger twigs without injuring the cambium?
11. How long will the fungus live in the bark of apple trees?
12. How may nurserymen avoid introducing the blotch fungus into the nursery?
13. In what manner is the disease probably most often introduced into young orchards?
14. How may the orchardist avoid introducing blotch into his new plantings?
15. What spray material has proved most satisfactory for apple-blotch control?
16. What phase of the disease does the most damage?

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### Late-blight of celery

Caused by *Septoria apii* (Br. and Cav.) Rostrup

This is one of the most cosmopolitan and troublesome of the several diseases attacking celery. It is variously referred to in the literature as leaf-spot, *Septoria* leaf-spot, *Septoria*-blight, leaf-blight, celery-blight and late-blight. The last name is preferable because it distinguishes this malady from the early-blight, another leaf-spot disease caused by a distinct species of imperfect fungus (*Cercospora apii*). In the United States this disease was first recorded in 1891 when it was mentioned in reports from three different experiment stations, namely, Delaware, Massachusetts and New Jersey. It had been reported in Italy the previous year which is the earliest known record of the trouble. Considerable damage from this disease was reported in central and western New York during the season of 1892. It was observed in California as early as 1895 and by 1897 had assumed serious proportions in that state. At the present time the disease is well distributed throughout the United States and in other countries where celery is grown. It is capable of causing great losses in localities where celery is grown intensively on large acreages. One of the best examples of serious loss caused by late-blight is that reported by Rogers (11) in California. A large celery-growing industry involving some 6000 acres sprang up on the peat lands in the vicinity of Smeltzer, California, and during the season of 1907-8 a loss of 1950 carloads valued at \$550,000 was caused by the late-blight



disease. It has been estimated that in 1915 the loss due to this trouble in the state of Michigan totaled at least \$1,000,000.

**Symptoms and signs.**— The characteristic symptoms consist of small dead spots scattered over the leaves and petioles. On the leaves

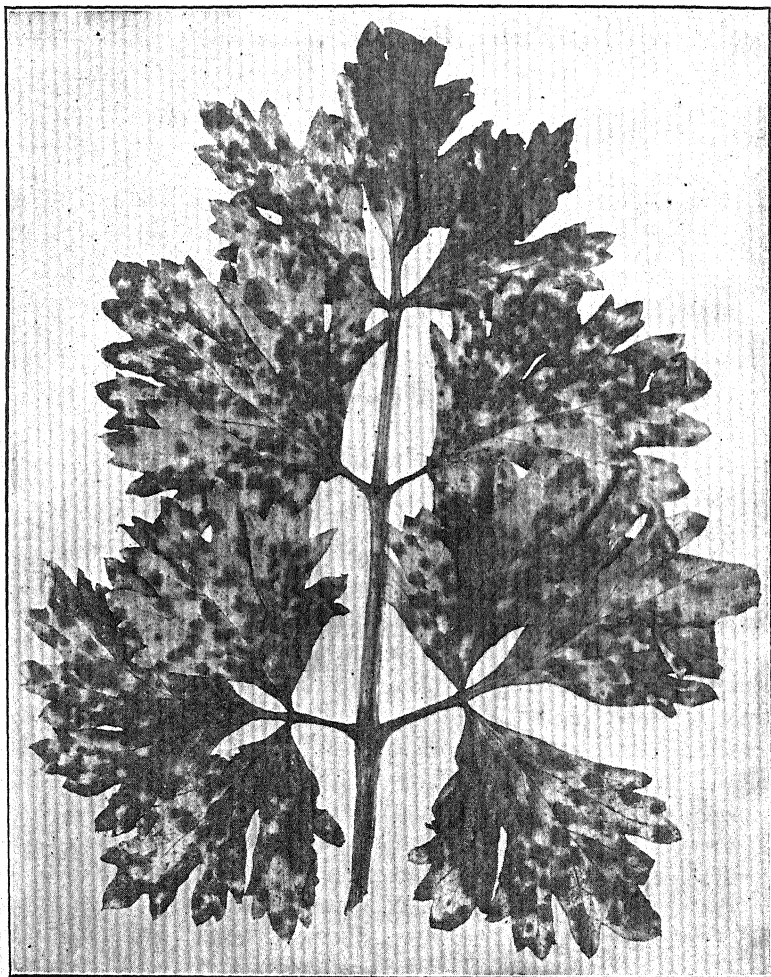


FIG. 167. — Celery leaf showing typical symptoms of late-blight.

these spots are more or less circular in outline and average one-sixteenth to one-eighth inch in diameter. At first the affected areas are light yellow in color, later turning brown or almost black. The lesions on the leaf stalks are more elongated in shape. Lesions may coalesce and form larger dead areas. The final diagnostic sign which distinguishes

this disease from all others on celery is the appearance of the minute black pycnidia on the dead lesions (Fig. 167). In typical cases these pycnidia are very numerous and are scattered densely over the dead areas. They can easily be seen with the naked eye.

**Morphology and life cycle of the fungus.** — This fungus has but one known spore form and belongs to the order of Fungi-Imperfecti which is characterized by the production of spores in the pycnidium type of fruiting body (Fig. 168). The ostiole of the pycnidium protrudes through the epidermis of the leaf and the spores are discharged in short

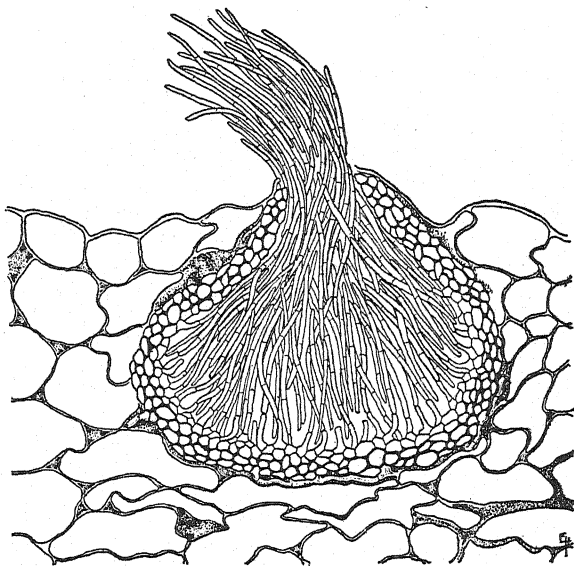


FIG. 168. — Section of pycnidium of *Septoria apii* showing the long, slender, septate spores.

cylindric tufts or bundles. The conidiospores are long, filiform, straight or slightly curved, septate by one to several cross walls, and measure 20–50  $\mu$  in length by 1–1.5  $\mu$  in width. The pycnidia are somewhat less than one-fourth millimeter in diameter. The fungus overwinters in the pycnidial stage on seeds or on diseased celery refuse left in the field. The fungus in the old debris rarely survives for longer than eight months to a year, but in the seed it may remain alive for more than two years. In the spring, spores are discharged from these overwintered pycnidia and infect the seedlings. The spores are discharged during wet periods and are disseminated in various ways. Wind and rain are the most important natural agencies while man undoubtedly disseminates spores

in the field by his cultural operations. In irrigated districts spores are probably carried along in the ditches and furrows. Of course the fungus may be widely disseminated on diseased seed. *Septoria apii* is a cool-weather fungus, thriving better at a temperature of 60° to 70° F. than at higher temperatures, hence in the temperate climates of the northern states it is apt to show up more abundantly in late summer and autumn as the plants approach maturity. In Florida where celery is planted in the fall and winter it is said that the *Septoria*-blight attacks the young plants at that season. The warm weather of the early spring checks the disease to some extent.

**Control.** — Based on the known facts in the life history of this disease there are three important items which should be considered in controlling it, namely, (a) the use of clean seed; (b) sanitation; and (c) spraying or dusting. Clean seed may be secured in various ways. Selection from disease-free localities or plantings is, of course, preferable if it can be done. Old seed is said to be free from the disease. Krout (8) found that 2 to 3 per cent of the conidia from pycnidia in the peduncles and pericarp of celery seed remained viable after 2 years but were all dead at the end of 3 years. Celery seed germinated well when 3 or 4 years old but seed more than 4 years old gave a poor germination test. Seed treatment with hot water or corrosive sublimate has been recommended. If mercuric chloride is used the seed should be soaked for 10 to 30 minutes in a 1-1000 solution of the chemical. The hot water treatment requires great care. Krout (8) found that the spores of the late-blight fungus are killed when heated for 30 minutes in water at 40° C. and the mycelium is killed at 45° C., while celery seed is not injured below 50° C. Above that temperature the seed is injured. Sanitation is necessary because of the fact that the fungus winters over in celery débris. The trimmings from diseased plants should not be left in the fields where celery is to be grown the next year nor near seed-beds. It is also a bad practice to throw this refuse on the compost heap unless it is to be left there for two or three years until completely rotted and all traces of the blight fungus are gone. Spraying is a highly recommended practice to protect the plants both from primary infections and from the secondary spread after the first crop of spores matures. Bordeaux mixture, 5-5-50, is commonly recommended. Spraying should begin when the plants are in the seed-bed and continue at intervals of a week or ten days until a few days before harvest. Dusting with 20-80 or 15-85 copper-lime dust has also proven effective. Rogers (11) recommends two applications of bordeaux spray to the seed beds before the seedlings are transplanted. After transplanting, the spraying should be resumed when the first blight appears or in any event not later

than six weeks after transplanting and repeated at intervals of one month until harvest, or if rains set in, at intervals of two weeks.

#### LABORATORY STUDY OF CELERY-BLIGHT

**A. Symptoms.** — Examine celery plants affected with late-blight. Note that the lesions occur on both blades and petioles of the leaves. Observe the number, size and color of the spots. Find the fruiting bodies of the fungus. On which side of the leaf do they appear? Are they numerous or scarce? What type of fruiting structure does this fungus produce? Are any general effects on the plant evident aside from the typical spotting? **Draw** to show typical symptoms.

**B. The fungus.** — Study sections cut through the leaf spots showing a sectional view of the pycnidia. Can you find the mycelium of the fungus in the leaf tissues? Note the bundles of spores protruding through the ostiole of the pycnidium. **Draw** to show the fungus, both mycelium and fruiting body, in place. **Draw** conidia enlarged.

*Life cycle.* — Look up the life history of this fungus. Where does it overwinter? How disseminated? What bearing does the method of hibernation have upon control measures?

#### REVIEW QUESTIONS

1. Describe the symptoms of celery late-blight.
2. Describe the morphology and life history of the fungus.
3. What does the age of celery seed have to do with control measures?
4. Discuss sanitary measures in relation to control of celery blight.
5. On what facts concerning the fungus is the recommendation of spraying based?

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### Bean Anthracnose

Caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. and Cav.

This disease has sometimes been ranked as the most important malady affecting beans in this country. Recent estimates of losses caused by it, however, have placed it a close second to the bacterial blight of this crop. It is probable that in some years it may still rank first in amount of damage done to the bean crop of the country. Other names that have been applied to the trouble are pod-spot, leaf-spot, rust and blight, the latter because of the frequent confusion of this disease with the true bacterial blight of beans.

**History and distribution.** — The disease was first described in 1878 by Saccardo (18). According to him it was first noted at Bonn, Germany, by Lindemuth. Later the specific name (*lindemuthianum*) was given to the fungus in his honor. The disease was reported from England in 1880. There is evidence (14) that it occurred in Massachusetts as early as 1882. By 1890 it had been observed in New York, Ohio, Michigan, Pennsylvania, Maryland, Louisiana, Wisconsin and Maine. Since then it has been reported from many European countries, including Ireland, Holland, Belgium, Denmark, Norway, Sweden, and Russia; from India, South Africa, Australia, New Zealand, several South American countries, Cuba, Alaska and Canada. At the present time there is scarcely a state in the Union from which the disease has not been reported at some time or other, and it probably occurs all over the world wherever beans are grown.

The disease has been the subject of extensive investigations during recent years. Barrus (5), in 1921, published a very complete résumé of the disease, including the results of extensive researches of his own on various phases of the problem, including physiological studies on spore germination and infection, susceptibility, and ecological studies. He also discovered two biologic strains of the fungus (4). Whetzel (21) published one of the earlier bulletins on this disease. Edgerton (12) in 1910 published the results of extensive investigations conducted in

Louisiana. Much of the recent work on this disease has dealt with the problems of perpetuation and control.

**Hosts and varietal susceptibility.** — The most susceptible host is the common bean, *Phaseolus vulgaris*, but the disease sometimes occurs on other species. It has been reported on Lima beans, Scarlet Runner, and cowpeas. However, it is not considered a serious disease of these other species. Some differences in varietal susceptibility among common beans have been found by different workers, but this work has not yet been carried far enough to be of any great value from the standpoint of control. One thing that has complicated the situation is the discovery of biologic strains of the anthracnose fungus. Barrus (4) has differentiated at least two strains of the fungus, to which all varieties of beans are not equally susceptible. Some breeding work has been done in an attempt to develop more resistant varieties (4, 16) with some degree of success.

**Economic importance.** — European writers mention epidemics of bean anthracnose, in various countries of that continent, in which serious losses were incurred. In the United States the disease is of great economic importance, especially in some of the leading bean growing states such as New York and Michigan. The disease was estimated (17) to have caused a loss of \$1,500,000 in Michigan during the year 1914 and a loss of twice that amount in 1915 in the same state. In 1915 the loss in New York was placed at \$700,000. The Plant Disease Bulletin (Supplement 24) places the loss in New York in 1921 at 4 per cent of the crop. The same publication (Supplement 30) estimates a loss of 117,000 bu. in Michigan in 1922, while in 1923 the loss in that state was estimated at 384,000 bu., or 5 per cent of the crop. In 1923 an estimated reduction in yield of 15 per cent was reported from Kentucky. In 1924 the reduction in yield in several states was estimated as follows: New York, 1.5 per cent; New Jersey, 2 per cent; West Virginia, 5 per cent; Michigan, 5 per cent; and Pennsylvania, 5 per cent.

The nature of the damage done is of several types. Badly diseased seeds germinate poorly sometimes resulting in poor stands. Diseased seedlings, even if they emerge from the ground, are not apt to develop into healthy, vigorous plants. A great reduction in yield may result from these two sources as well as from later infection on the pods. In green beans severe spotting of the pods renders them unsalable.

**Symptoms.** — The disease commonly affects all parts of the plant above ground and may sometimes be found on the roots also. The pods, seeds, leaves and stems show characteristic symptoms, but the most pronounced and most universally observed effects of the disease are the very conspicuous spots occurring on the pods.

*Pod symptoms.* — The first noticeable symptoms on the pods are minute brown specks appearing on the surface of the ovary wall. These spots rapidly enlarge and become darker in color finally appearing almost black at the center with a lighter border (Fig. 169). The larger lesions reach a diameter of one centimeter or more with more extensive areas



FIG. 169. — Bean anthracnose. Typical lesions on the pods. (Photograph by Bailey, Ore. Agr. Exp. Sta.)

formed by the coalescence of two or more spots. Individual cankers are usually more or less round or oval in shape. As the lesion develops the surface becomes sunken. The fungus may penetrate the wall of the pod and enter the seed. The final signs appear on the surface of the affected area with the development of the spores of the organism. The fruiting pustules (acervuli) break out in large numbers over the surface



of the canker and under moist conditions the spores ooze out in enormous numbers. These spore masses are pinkish or flesh-colored and sometimes are so numerous as to cover the whole surface of the canker and mask its dark color. Under drier conditions the spore masses dry down and lose their pink color becoming gray or darker in color. Anthracnose

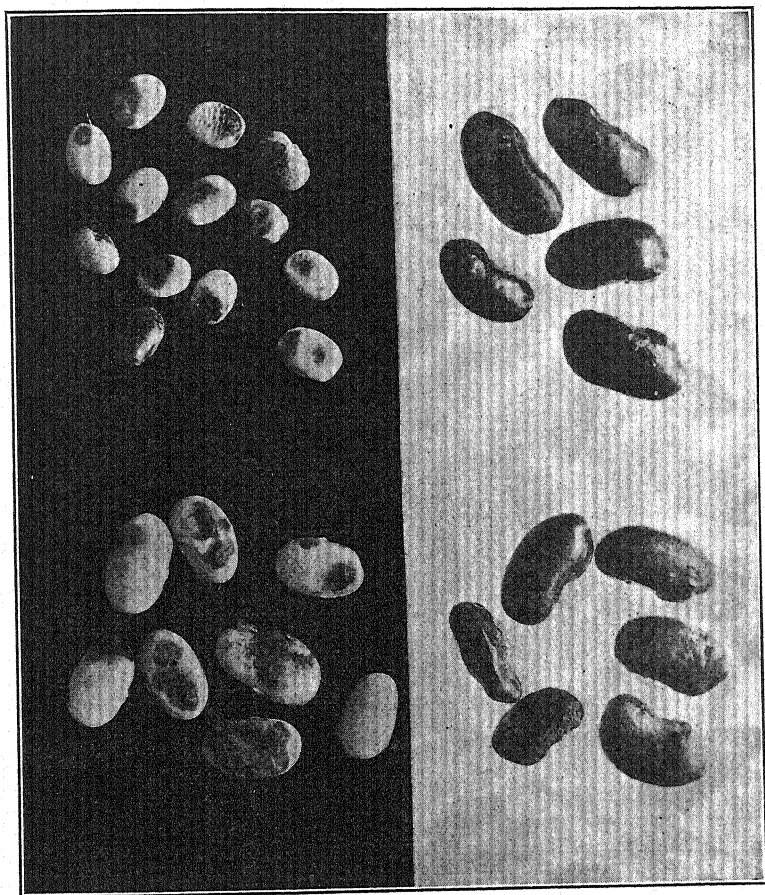


FIG. 170. — Anthracnose on bean seeds. Upper left, Navy Pea; upper right, Red Kidney; lower left, White Marrow; lower right, Brocton. (After Barrus, Courtesy Cornell Univ. Agr. Exp. Sta.)

spots are sometimes confused with lesions of the bacterial blight on bean pods. They may usually be distinguished, however, by the more definite and regular outline and sunken surface of the former. The fruiting pustules of the anthracnose fungus also will serve to distinguish



it from the blight disease which exudes only a yellowish bacterial ooze over the surface of the spots. (See under Bean Blight, p. 200.)

*On seeds and seedlings.* — When the fungus penetrates through the pod into the seed, the latter are discolored more or less according to the severity of the attack (Fig. 170). Slight cases may be scarcely noticeable while in other cases conspicuous spots, yellow, tan, or blackened

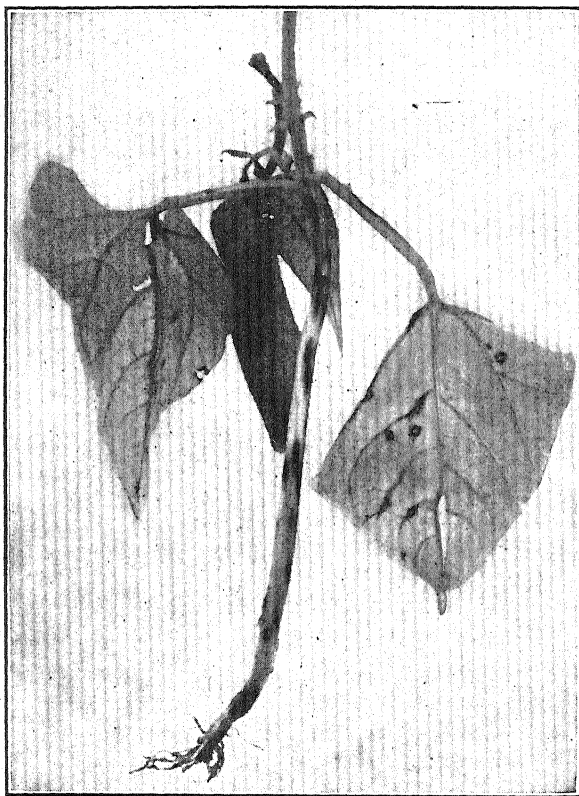


FIG. 171. — Bean anthracnose lesions on stem and leaves. (After Whetzel, Courtesy Cornell Univ. Agr. Exp. Sta.)

and sunken, may appear. The spots are more noticeable, of course, on white varieties of beans than on colored varieties. Badly diseased seed may not germinate. If seedlings from diseased seed do appear above ground they may show symptoms of serious infection. Large dark cankers may appear on the cotyledons or on the young stem. These cotyledon lesions resemble those on the pods and may produce spores which serve as a source of danger to other plants.

*On leaves and stems.* — The lesions on the leaves occur mainly along the veins and petioles, and appear usually on the under side of the leaf (Fig. 171). Small parts of the leaf blade surrounding the vein lesions are killed giving rise to elongated, irregular-shaped dead areas. On the stems elongated dark red or blackened cankers are produced. On young stems a rotting may accompany these lesions while older stems may become cracked.

**Morphology and life cycle of the fungus.** — The mycelium penetrates the invaded tissues in great profusion (Fig. 172). The hyphae enter the cells and may fill them completely. The young hyphae are hyaline but with age the mycelium becomes darker in color. When the invaded tissues have been killed there are formed dense stromata of fungus tissue

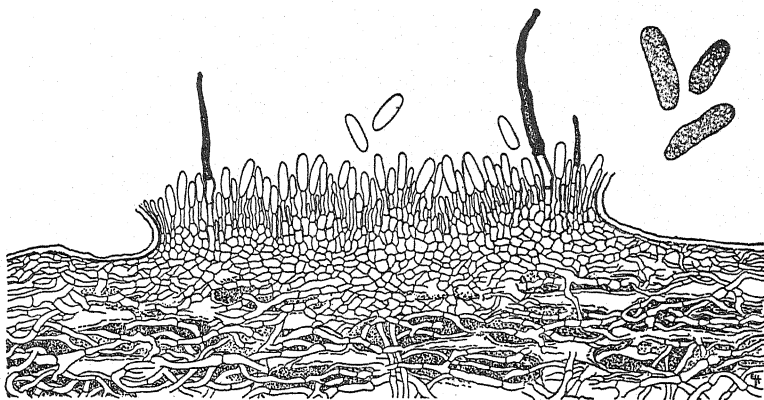


FIG. 172. — Section of acervulus of *Colletotrichum lindemuthianum*. At upper right, conidiospores enlarged.

in numerous places in the epidermis and just beneath the cuticle. From these stromatic cushions the spores are produced. Only one spore stage is certainly known in the life history of this fungus, namely, the imperfect or conidial stage. An ascigerous stage has been described (19) in cultures, but lack of sufficient confirmation of the occurrence of this stage in nature has led some pathologists tentatively to retain this fungus among the Fungi-Imperfecti. The imperfect fruiting body is a typical acervulus or saucer shaped structure with its mass of simple conidiophores breaking out through the cuticle on the surface of the cankers (Fig. 172). The conidiophores are erect, unbranched hyphae, measuring about 45 to 55  $\mu$  in length by 4 to 5  $\mu$  in diameter. Scattered among the conidiophores, frequently around the margin of the pustule, are long, dark colored setae, which protrude far beyond the

ends of the conidiophores, ranging up to  $90\ \mu$  long (Fig. 172). The conidia are produced at the apex of the conidiophores. Many conidiospores may be produced in succession from a single conidiophore. The spores are oblong with rounded ends, usually straight but sometimes slightly curved, and measure  $13\text{--}32 \times 3.5\text{--}5\ \mu$ . They are hyaline and somewhat guttulate. They ooze from the acervuli in mucilaginous masses and thus massed have a pinkish color.

*Overwintering.* — The chief means of perpetuation and overwintering of the bean anthracnose fungus is in the form of mycelium in diseased seeds, although it may overwinter in other ways to a certain extent. When seeds bearing the mycelium are planted the soil moisture stimulates the fungus to renewed growth. If the vitality of the seed is not impaired too much the seedling may emerge, but by this time lesions will have developed on the cotyledons or other parts of the seedling sufficiently to produce spores. Under proper conditions these spores may cause infection on neighboring plants and thus the life cycle is completed. There is some evidence (5) that a few spores may survive the winter in the soil, but apparently they do not constitute an important source of infection the following spring. It is known to live over winter in diseased bean débris left in the field.

*Dissemination and infection.* — Since the spores are held together in gelatinous masses, moisture is necessary to dissolve the mucilaginous substance and thus make dissemination possible. The spores are largely disseminated by rain and are probably widely scattered by cultural operations when the plants are wet with dew or rain. Insects probably disseminate spores to some extent. Temperature and humidity play an important part in the germination of the spores and the subsequent development of the fungus. The optimum temperature for the growth of this fungus is about  $22^{\circ}\text{C}$ . ( $71^{\circ}\text{F}$ .), and the maximum is around  $34^{\circ}\text{C}$ . ( $93^{\circ}\text{F}$ .). Edgerton (12) states that in Louisiana the anthracnose fungus is of no consequence in the field during the long hot summers, although it is prevalent during the cooler part of the season. Moisture is necessary for infection to occur. Lauritzen (15) determined that a film of moisture on the leaf surface is not absolutely necessary, but that infection could take place at humidities of 95.8 per cent when the temperature was kept at an optimum degree.

*Control.* — The life history facts that must be considered in controlling this disease are: (a) the fungus overwinters in seed, and also in bean refuse in the field; and (b) it is scattered about the field by rain, cultural operations and possibly insects. Consequently control measures will include: (a) the use of clean seed; (b) crop rotation; (c) sanitation; and (d) the consideration of the prevention of field spread by means of

sprays. No satisfactory seed treatment has been devised because the fungus is imbedded in the seed. Selection of clean seed is the chief preventive. This can be done better by securing seed from disease-free fields rather than by attempting to select clean seed from beans containing some disease. Nearly always some communities can be found where the disease is absent. Crop rotation and sanitation are directed toward the control of any outbreak of the disease that might come about through wintering over on bean debris in the field. Spraying with bordeaux to prevent the spread of the disease in the field has given fairly good control.

The use of resistant varieties, of course, is always a possibility that should be carefully considered in trying to control plant diseases. As indicated previously, some differences in varietal susceptibility have been found and work on this problem will undoubtedly be continued. Barrus (4) found five varieties that showed more or less resistance to both strains of the fungus which he differentiated. Of these five, the Red Kidney bean is mentioned as the most promising.

#### LABORATORY STUDY OF BEAN ANTHRACNOSE

**A. Symptoms.** — All parts of the bean plant are subject to attack by this disease. Examine pods and seeds showing the effects of the disease. On the pods note the sunken canker-like spots. Observe the size, shape and color. On the surface of the canker spots note the fruiting pustules of the fungus. Compare the pod spots caused by this fungus with the pod spots caused by the bacterial blight organism. What are the distinguishing marks? Observe the symptoms on the seeds. Compare the two diseases as to effects on the seeds.

Now examine plants showing the effects of the disease on the cotyledons, stems and leaves. Make drawings to illustrate all symptoms found on pods, seeds or other parts of the plant.

**B The fungus.** — Examine sections cut through a typical canker spot on the pod where the fungus is sporulating. Look for the mycelium in the diseased tissues. Select a good section through an acervulus and examine with the high power objective of the microscope. Find conidiophores, conidia and setae. Draw a sectional view of the acervulus. Also draw a single conidiophore and a few conidia much enlarged.

A perfect stage has been described for this fungus but it is apparently rare and is usually not available for study.

**Life cycle.** — Look up the life history of this fungus, and find out how it overwinters and its usual method of dissemination.

**C. Notes.** — Write complete notes on this disease, covering symptoms, life history and control in detail.

#### REVIEW QUESTIONS

1. Describe the symptoms of bean anthracnose. Distinguish from the bacterial blight of beans.

2. Describe the complete life cycle of the bean-anthrachnose fungus. Give particular attention to the methods of perpetuation and dissemination.

3. How does the manner of overwintering compare with that of bean blight? What effect has this upon the control measures for the two diseases?

4. Is it possible to select seed free from anthracnose in a field where much disease exists? How? Compare with bacterial blight in this respect.

5. What is the value of spraying as a control for this disease? Outline a spray schedule for bean anthracnose. Give your reasons for arranging the schedule as you have.

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### Peach-scab

Caused by *Cladosporium carpophilum* Thüm.

This disease is one of the most common and widespread maladies of the peach in the eastern half of the United States. It has been called freckles, black-spot and peach-scab. The first record of this disease was made by Von Thümen (12), who reported it from Austria in 1877. He described the causal fungus and gave it the name by which it is still known. There is proof that specimens affected with this disease were collected in this country as early as 1881. The trouble first received pathological mention in the United States in 1889 when Arthur (1) described the disease on peach fruits in a bulletin of the Indiana Experiment Station. In 1904 Clinton (4) reported scab injuries on the fruits, twigs, and leaves of the peach. It was not until more recently, however, that the lesions of fruits, leaves, and twigs were all experimentally demonstrated to be due to the same pathogene. Between 1895 and 1915 considerable attention was given to the control of the disease by spraying. Selby (10), as a result of seven years of experimentation, demonstrated that the scab of peach can be controlled by bordeaux sprays, but this fungicide is not generally popular as a spray for peach trees, because of the injury caused to the foliage. Scott and his associates (7, 8, 9), during the years 1907 to 1911, used self-boiled lime-sulfur for peach-scab control with satisfactory results. However, it was not until 1917 that anything but fragmentary information on this disorder of peaches was available. In that year Keitt (6) published a very complete treatise on peach-scab, including a discussion of the taxonomy, morphology, and physiology of the causal organism, the pathological anatomy, ecological relations, and the life cycle of the pathogene.

The disease is now quite widely distributed in this country occurring in most of the states as far west as Nebraska, Colorado, and Texas. Outside of the United States the disease is known to occur in Austria, Holland and other European countries, in Australia, South Africa and Canada. It does not seem to have received a great amount of attention in any of these foreign countries.

**Hosts and varietal susceptibility.** — This fungus attacks the peach, plum, apricot, cherry and nectarine, but it is primarily a parasite of the peach. Little damage has been reported on fruits other than the peach. Some peach varieties are more susceptible than others. Late varieties are more apt to be severely attacked than early maturing varieties. From available information the following list will indicate in a general way the varying degrees of susceptibility of different varieties. Of

course, the ranking of any variety will vary more or less with the locality and climatic conditions, and from season to season.

*Very susceptible.* — Mountain rose, Reeves, Salway, Bilyew, Rivers, Tennessee, and Heath.

*Moderately susceptible.* — Belle, Elberta, Late Crawford, Alexander, and Edgemont.

*Slightly susceptible.* — Hale Early, Hiley, Early Crawford, St. Johns, and Champion.

**Economic importance.** — Although this fungus attacks the twigs and leaves as well as the fruits the chief damage sustained is due to the effects on the fruits. While the fruit is not destroyed directly by the scab fungus, its quality, appearance and salability are affected very adversely by the occurrence of any considerable amount of this disease. In 1910 Scott and Ayres (8) estimated that peach-scab was causing a decrease of 25 per cent in the market price of the fruit in some of the eastern states. In 1922 The Plant Disease Reporter (Supplement 30) gave the estimated loss from peach-scab in some of the chief peach growing states as follows: Georgia, 3 per cent or 170,000 bu.; New York, 63,000 bu.; Arkansas 77,000 bu.; and for the whole United States, 3.3 per cent, or, 2,326,000 bu. In 1924, The Reporter (Supplement 43) estimated the losses in North Carolina at 3 per cent or 75,000 bu.; Georgia, 191,000 bu.; Texas, 102,000 bu.; Arkansas, 97,000 bu.; and in the entire United States, 707,000 bu.

**Symptoms.** — The symptoms of this disease appear on the fruits, leaves and twigs. The most striking symptoms and those which do the most harm are found on the fruits. Those on the twigs and leaves are less noticeable and are of little economic importance as far as direct damage is concerned.

*On the fruit.* — The evidences of well-developed scab infections on peach fruits consist of small, circular, olivaceous or black spots on the surface of the fruit (Fig. 173). When first noticeable these spots are very small, less than half a millimeter in diameter, and greenish or olivaceous in color. The spots gradually enlarge until they become 2 to 5 mm. in diameter. Sometimes they are so numerous that they coalesce so that one whole side of the fruit may be covered with a solid scab spot. The lesions are always on one side of the fruit, that is, the side most exposed to the elements and on which the hairs gradually are worn off so that the surface is more easily wettable. The protected side is not easily wetted and consequently infection does not readily occur on that surface. The fungus does not penetrate deeply, therefore the spots are confined to the skin of the fruit. However, when fruits are infected while still very immature, the infected surface, which becomes corky,

has a tendency to crack as the fruit matures. Very deep cracks reaching to the pit sometimes occur. Such breaks in the skin of the fruit offer easy access to brown-rot or other fungi. On badly scabbed fruits the scabby side does not ripen uniformly with the clean side of the peach, so that in using such fruit only the clean side is suitable for eating, and the scabby side is usually worthless.

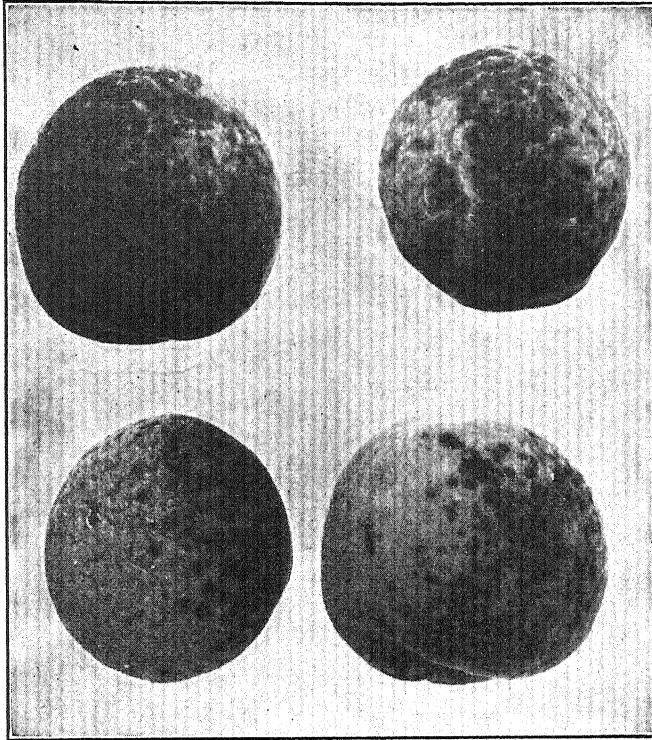


FIG. 173. — Peach-scab on half-grown fruits.

*On the leaves.* — The symptoms on the leaves seldom amount to more than a speckling of a part of the leaf surface. The spots are small, rarely more than 1 or 2 mm. in diameter and are relatively inconspicuous. The first symptoms evident to the naked eye occur as minute, indistinct discolored areas on the under surface of the leaf. The slight discoloration becomes intensified to a light brown, sometimes with a tinge of pink or purple. Later the effects become evident on the upper surface as pale yellow or purplish areas. The conidiophores of the fungus finally appear over the surface of the spots on the underside of the leaf, giving a somewhat olivaceous color.



*On the twigs.* — The twig lesions always appear on the young current season's growth. They are usually oval in shape and vary in size, measuring up to 5 by 8 mm. in diameter. The spots are at first indistinctly defined, but later become brown in color with slightly raised, dark-brown or purplish borders (Fig. 174). No part of the lesion ever becomes more than slightly elevated below the surrounding surface.

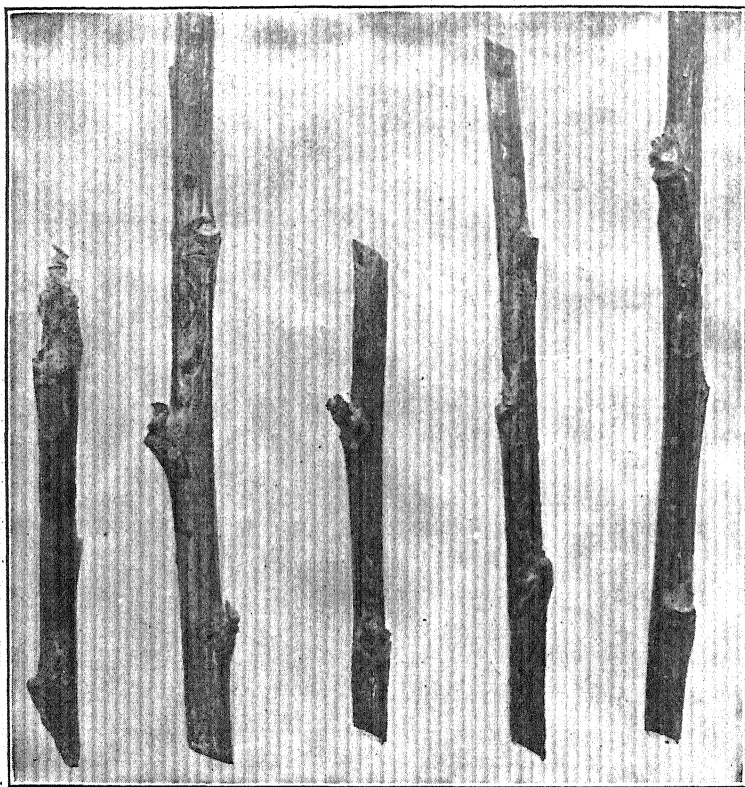


FIG. 174. — Peach-scab lesions on twigs. (Photograph by M. W. Gardner, Purdue Agr. Exp. Sta.)

The infection extends only a few cell-layers deep and seldom reaches the cambium so that very little damage is done. During the spring of the following year the fungus fruits abundantly on the surface of the lesions, the dark-colored conidiophores and conidia giving the year-old cankers an olivaceous coloration. During the second and third years the fungus dies out and the further growth of the twigs gradually obliterates the cankers.

**Morphology and life history of the fungus.**—The mycelium is at first hyaline, but later becomes dark in color. On the fruit the hyphae mass in irregular pseudoparenchymatous clumps over and among the surface cells of the fruit (Fig. 175). Stromata similar in nature also form beneath the cuticle on twigs. From these masses of fungus tissue the conidiophores arise. These are short, dark-brown hyphae, more or less flexuous, septate by one or more cross-walls, and seldom branched (Fig. 175). The conidia are produced at the apex of the conidiophores usually one at a time, but under certain conditions they may appear in short chains. Mature conidiospores are somewhat ellipsoid to fusoid-ovate in shape. They may be either single celled or once-septate (Fig. 175). They vary considerably in shape and size. The average measurements run about  $15 \times 5 \mu$ . No other spore form is known for this fungus.

The fungus overwinters as mycelium in the stem cankers. Abundant conidia are produced in the spring on the surface of these lesions and serve as the source of primary infections on fruits, twigs and leaves. The fungus has a rather long incubation period and because of the fact that very young peach fruits are very woolly the first symptoms of infection appear rather late in the season. Usually few infections are observable until 5 to 7 weeks after the petals fall. On account of this lateness in the appearance of the disease, infections from secondary conidia are of little consequence until very late in the season. An abundance of moisture during the spring and early summer and a long growing season favor the inception and development of the disease.

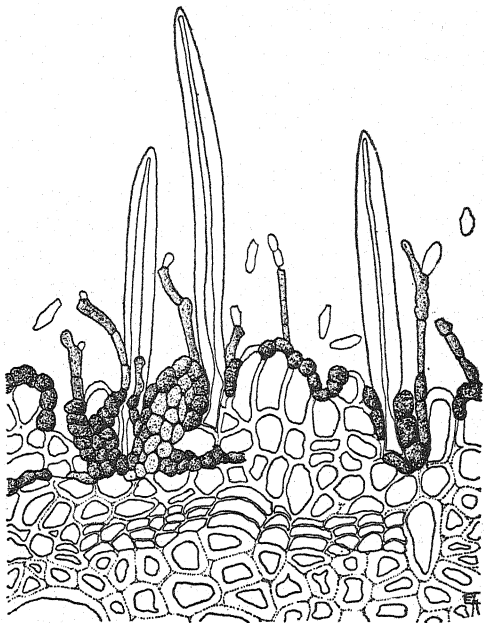


FIG. 175. — Section of the skin of a peach fruit made through a scab spot, showing the groups of dark colored fungus cells from which the conidiophores arise, and also the long, spike-like hairs which cover the surface of the peach.

**Control.** — In view of the life-history facts just stated, it is evident that only two possibilities are available in devising control measures for peach-scab. These are: (a) getting rid of the source of primary inoculum which consists of conidia from the overwintered cankers, and (b) the application of a protective spray. The first possibility is eliminated immediately because of the numerous and inconspicuous lesions scattered all over the tree. It would manifestly be impossible to eliminate all of them without cutting out every bit of one-year-old wood on the tree. Consequently the only alternative left is the use of sprays. The early studies on the control of this disease indicated that bordeaux mixture would control the disease but in most localities too much spray injury results from the use of this material to permit of its use on peach foliage. The more highly concentrated forms of lime-sulfur are also harmful. It has been shown that self-boiled lime-sulfur, 8-8-50, or wettable sulfur, 5 lbs. to 50 gals. of water, will control the disease and at the same time are safe to use on peach trees. The schedule varies for early and later varieties of peaches. For early varieties one application made about one month after the petals fall is sufficient. For mid-season varieties two applications are advisable, applied one month after petal-fall and three weeks later respectively. Late-maturing varieties require three applications, the first two as above and a third applied about one month after the second.

#### LABORATORY STUDY OF PEACH-SCAB

**A. Symptoms.** — Examine fruits, leaves and twigs of peach affected with the scab disease. On the fruit note the color and extent of the infected areas. Compare with apple scab as it appears on apple fruits. Is there any similarity? How do the two diseases differ? When a large number of the peach-scab lesions coalesce what is the general effect on the fruit, especially in advanced stages of the disease? Describe the symptoms as seen on leaves. Examine peach twigs showing scab lesions. On what aged twigs can scab be found? Note the size and color of the lesions. How deep does the fungus penetrate into the bark? Compare these peach-scab twig lesions with the twig cankers of apple blotch. Draw to show all symptoms.

**B. The fungus.** — Examine sections cut through a typical scab spot on the skin of peach fruit. Note the clumps of dark-colored mycelium at the surface and the conidiophores and conidia arising from these masses of mycelium. Be careful to distinguish the fungus from the numerous plant hairs arising from the epidermis of the fruit. Draw a portion of the section to show the fungus and its fruiting structures. Likewise examine sections cut through the twig lesions. Does the fungus sporulate on the twigs as well as on the fruit? Where and in what form does the fungus overwinter? What is the source of inoculum for the primary infections? Has the fungus a perfect stage?

**C. Notes.** — Write complete notes covering especially symptoms, cause and control.

## REVIEW QUESTIONS

1. Describe the symptoms of peach-scab on fruit, twigs and leaves.
2. How many spore forms appear in the life cycle of this fungus? Describe the life history.
3. Why do young peach fruits so readily escape infection until later in the season?
4. What weather conditions are favorable for infection and development of the disease?
5. What is the known geographical distribution of this disease in the United States?
6. What fruits besides peaches are susceptible to this disease?
7. What is the nature of the damage done by peach-scab?
8. How deeply does the fungus penetrate into the twigs?
9. Look up the methods and results of cultural and inoculation work as described in reference 6.
10. Discuss control methods.

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## Common Scab of Potatoes

Caused by *Actinomyces scabies* (Thax.) Güssow

Some confusion has existed as to the correct common name which should be applied to this disease. There are two other potato diseases which may be confused with it, namely, powdery scab and Rhizoctonia,

the latter sometimes called black scurf or scab. In Europe the powdery scab is much more common than in America, and in the early European writings, before the cause of either was well known, the powdery scab was not clearly distinguished from the common scab. In America the common potato scab disease has been variously called American scab, *Oöspora* scab, corky scab, and common scab. While there is some difference of opinion as to the most suitable common name for this disease the most commonly used name in American literature is "common scab."

**Historical.** — The original home of the potato scab organism is not known. At present it seems to be found in the soil quite generally. The disease as it occurs in potatoes has been known in Europe and America for a long time. It might have been introduced into Europe from South America on the first importations of potatoes. However, the first published description of the disease dates back to 1825. Between the years 1840 and 1890 several European writers advanced theories as to the cause of the disease but because of the fact that they did not distinguish between the different kinds of scab their writings are confusing. In 1886 Brunchorst (6) described the causal organism of powdery scab but it remained for Americans to show that there are different kinds of scab (12) and to determine the true cause of the common scab as distinguished from the powdery scab. Thaxter (22) was the first to discover the organism causing common scab. In 1891 he described a fungus-like organism which he isolated from scabby potatoes and with which he was able to produce the disease by artificial inoculations. He named the organism *Oöspora scabies*. Later Güssow (11) changed this name to *Actinomyces scabies* which name the organism still bears. Since the discovery of the cause of the disease the attention of plant pathologists has been directed more particularly to the problem of control by seed treatment and toward discovering the soil reactions and other ecological factors which favor or retard the development of the disease.

**Hosts and varietal susceptibility.** — While the potato is the most extensively attacked plant it is by no means the only one. Beets, turnips, mangels, rutabagas, parsnips, radishes, and carrots are affected occasionally. Not all members of the family to which the potato belongs, the Solanaceae, are subject to attack, the tomato, tobacco and pepper being immune. Most writers on the subject are agreed that not all potato varieties are equally susceptible. Some investigators merely list certain varieties as susceptible and others less so while other workers have tried to correlate certain tuber characteristics with scab resistance. In 1906 and 1907 Stewart (21) made extensive observations on varietal

resistance to scab in Vermont. He states that of the 74 varieties which he used in these experiments, none were immune, although not all were equally susceptible. He was not able to definitely correlate differences in susceptibility with skin or other characters in the tubers. Lutman (14), on the other hand, claimed to find a marked relation between the thickness of the skin on tuber varieties and susceptibility to scab. He found the true russet types of tubers markedly resistant and the white, thin-skinned varieties most susceptible, while the semi-russets were intermediate in susceptibility. Following are some of the varieties listed by Lutman, in his 1914 trials, in order of their freedom from scab: Cambridge Russet, Scab Proof, Burbank's Russet, Dakota Red, Million Dollar, White Ohio, Rural New Yorker No. 2, Irish Cobbler, Early Rose, Vermont Gold Coin, Early Ohio and Triumph. In subsequent trials the order of susceptibility shifted somewhat but he was still able to draw the general conclusion that russet types are more resistant than other types.

**Economic importance.** — In estimating the loss caused by common potato-scab several items must be taken into consideration. (a) There is probably an actual reduction in yield where scab infection is very abundant. (b) Blemished tubers depreciate in market price. (c) The consumer loses by reason of the thicker paring necessary to remove all scabby tissue. (d) Additional losses are caused by invasion of scabbed tubers by secondary organisms. The greatest loss from scab undoubtedly results from depreciation in the market value of the crop, severe cases even rendering potatoes unsalable. The estimated percentage of loss (2) in 1925 in several states follows: New York, 7; Kansas, 10; Virginia, 5; Iowa, 5; New Jersey, 4; and Minnesota, 1. In 1924 the estimated percentages (1) in a few states were: New York, Iowa and Michigan, 5; North Carolina, Kansas and New Mexico, 3; New Jersey, Pennsylvania and Arizona, 2; Illinois, Alabama and Texas, 1 per cent.

**Symptoms.** — The scab lesions occur only on the underground parts of the potato plant, chiefly on the tubers. There are no symptoms apparent above ground therefore the disease cannot be detected until the tubers are dug. The first noticeable symptom is a minute brown spot on the skin of the developing tuber. These spots rapidly increase in size until they are several millimeters in diameter (Fig. 176). If the spots are numerous they may coalesce more or less forming still larger spots. Sometimes the entire surface of a tuber may become scabby. As the spot enlarges cork cells are formed. These cork layers become several cells thick and broken or roughened on the surface giving the characteristic appearance which the name, scab, suggests. In some cases the scab lesion may penetrate to the depth of a half centimeter or

more. The corky surface of the scab usually becomes cracked and furrowed, frequently in a more or less concentric manner. Some writers have attempted to distinguish several different types of symptoms, as, shallow scab, deep scab and bulging scab. Other terms suggested are warty scab, surface scab and pitted scab. These types are not hard and fast but grade into each other and several different types may sometimes be found on a single tuber.

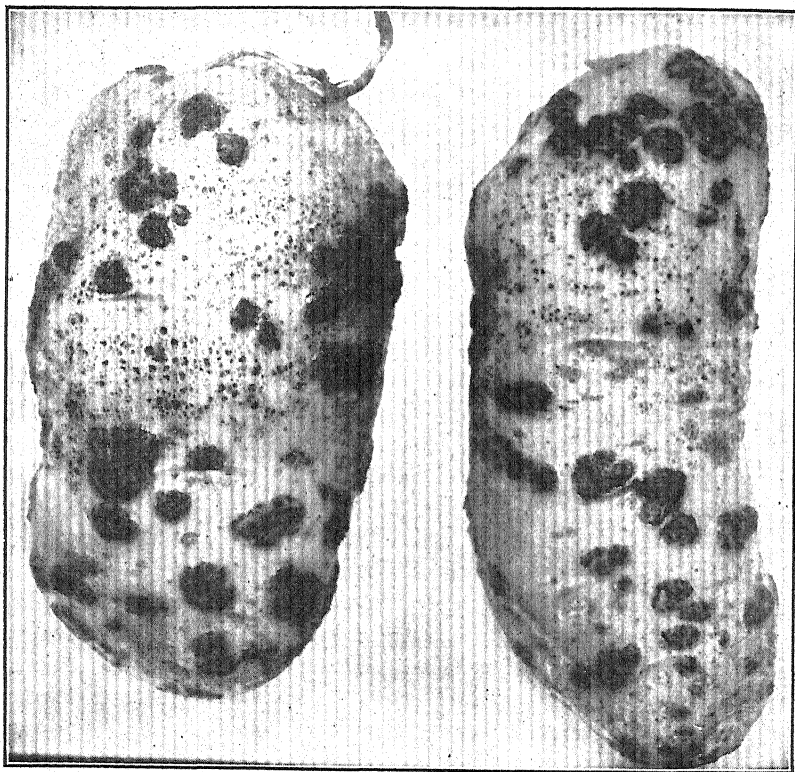


FIG. 176. — Potato tubers affected with common scab. (After Bailey, Ore. Agr. Exp. Sta. Crop Pest Rept., 1911-12.)

**Morphology and life history of the causal organism.** — At various times since the discovery of the common scab organism doubt has been expressed by different writers as to the nature and proper classification of this pathogene. Since Güssow transferred it from the genus *Oöspora* to the genus *Actinomyces*, most writers have referred to the organism as one of the thread bacteria. More recently Drechsler (9) has demonstrated convincingly that it is a fungus rather than a bacterium and



belongs in the Fungi-Imperfecti. Many species of the genus *Actinomyces* have been isolated from soil so that it is not at all surprising that the potato scab species is found to be a common inhabitant of many soils. The vegetative structure consists of threads or filaments, branched, wavy, irregularly segmented, and ranging from .5 to 1  $\mu$  in diameter. The filaments break up into short cells called gonidia or conidia which function in reproduction (Fig. 177). Cultures on agar plates form roundish colonies, with irregular margins, raised, rugose surface, and gray to buff or medium brown in color. The organism hibernates on potato tubers and in the soil. It seems able to exist indefinitely on humus in the soil and since it also attacks the roots of some other plants besides potatoes, it is frequently encountered in soils which have never grown potatoes. Infection occurs mainly through stomata or young lenticels

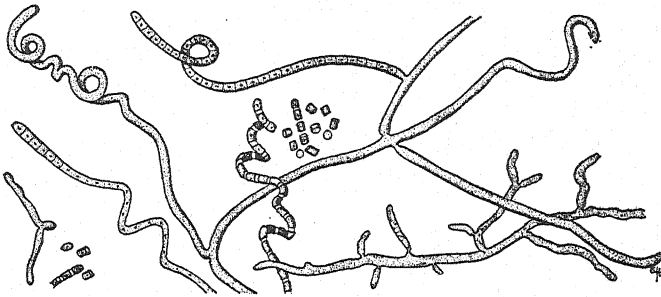


FIG. 177. — Mycelium and conidia of *Actinomyces scabies*. (Redrawn after H. Fellows, from unpublished manuscript.)

(10) at the apical end of the growing tuber, never after the tuber ceases growth nor after the normal cork layer is well developed. Scabs occurring at the base of a tuber are the result of infection which took place when the tuber was very small.

*Environmental factors.* — It has long been known that an alkaline condition of the soil favors scab infection and development. Many growers have noted the fact that ashes, lime or fresh barnyard manure applied to the soil before planting potatoes resulted in increased amounts of scab. This fact has led to the conviction on the part of farmers that these substances cause scab, whereas the true explanation is that these applications favor the development of the scab organism by bringing about a soil reaction favorable to the growth of *Actinomyces*. Other important factors are the temperature and the soil moisture content. It has been determined (12) that a soil temperature of 22° C. is favorable for both tuber and scab development. Sanford (18), in Alberta, Canada, found that scab developed more abundantly in a dry soil than in a moist



soil. At the same time he found that, in his experiments, the dry soil was more acid than the wet soil. This would seem to contradict the idea that alkalinity is the sole determining factor in scab development. As our knowledge of the relation of ecological factors to plant diseases increases it becomes more and more evident that epidemics do not depend upon a single factor but rather upon a combination of soil, temperature and moisture factors. (See Chapter V.) In the case of potato scab, as nearly as can be determined from available data, it would seem that a certain combination of dry weather, moderate temperature and a soil reaction not far from neutral in either direction is conducive to bad scab infection. However, there are still confusing instances of apparent contradictions in regard to the influence of these environmental factors. In 1924 some collaborators for The Plant Disease Survey (1) turned in conflicting reports. Scab was reported as severe in Kansas on account of dry weather, while New York and Indiana reports indicated severe scab infection due to wet weather. No doubt this discrepancy could be explained if all other factors could be taken into consideration.

**Control.** — In considering control measures for common potato scab two factors must be kept in mind: (a) the fact that scabbed tubers carry the organism, and (b) the propagation indefinitely of the organism in the soil humus. The grower should make every effort to secure scab-free seed potatoes for planting. If the home grown seed is not sufficiently clean, certified seed should be purchased. Since even certified seed is not necessarily free from scab, seed disinfection should always be practiced. There are two fungicides which have been effectively used against scab, namely, formaldehyde and corrosive sublimate. If formaldehyde is used the tubers should be soaked for two hours in a solution of 1 pint of 40 per cent formaldehyde to 30 gal. of water. It is customary to recommend the use of corrosive sublimate instead of formaldehyde because the former is more effective for *Rhizoctonia*, another potato disease which is usually present, and thus treatment for both diseases can be performed at the same time (See under potato *Rhizoctonia*, p. 426). The formula for corrosive sublimate (bichloride of mercury) treatment calls for 4 oz. of corrosive sublimate in 30 gal. of water. An earthenware or wooden vessel must be used because the solution attacks metal chemically. The tubers should be treated in this solution for about one and one-half hours. This solution is very poisonous therefore due care should be taken to protect children and animals. Recently hot formaldehyde has replaced the above treatments to some extent for both scab and *Rhizoctonia* (See p. 427).

Equally important as the use of clean seed is the practice of crop

rotation. If potatoes are grown on the same ground year after year and any scab occurs in the crop there is a tendency for the scab organism to increase in the soil and thus make it increasingly difficult to grow a scab-free crop even if the best methods of seed selection and disinfection are used. The rotation of crops in such manner as to grow potatoes in the same field not oftener than once in every four or five years is a good practice for various reasons and it should be effective in reducing the amount of the scab organism in the soil, although it is not contended that it will rid a soil completely of this organism since it is known to occur in some virgin soils which have never grown potatoes.

Since the organism is apt to be present in the soil, the use of clean seed and crop rotation alone will not always insure a clean crop. The problem of soil treatment to reduce the prevalence of scab involves the manipulation of the environmental factors in such manner as to bring about conditions unfavorable for the growth of the scab organism. Of the three factors discussed previously, soil reaction, temperature, and moisture, the one that has received most attention is the reaction of the soil. Temperature, of course, cannot be controlled, and moisture likewise except in irrigated sections. Since an alkaline condition favors scab, any treatment that will increase the acidity of scab-infested soil will tend to decrease the amount of scab. The addition of lime or alkaline fertilizers immediately previous to planting a crop of potatoes is inadvisable. However lime is beneficial to many crops therefore the crop rotation will have to be considered in this connection. In case the soil needs lime it should be added preceding some other crop rather than the potato crop. Where the potato alone is considered the use of acid fertilizers and of sulfur is sometimes advisable. A great deal of experimentation on the use of sulfur as a scab control has been carried out. The principle involved is based upon the fact that the oxidation of the sulfur increases the soil acidity. Ordinary sulfur may be used but in many cases inoculated sulfur is better because the sulfofying bacteria are not abundant in all soils, hence the addition of the oxidizing bacteria to the sulfur previous to its application to the soil will hasten the bringing about of an acid condition. The sulfur may be applied at rates of 200 to 600 lbs. per acre depending upon the severity of scab in the preceding crop. If a nitrogen fertilizer is needed ammonium sulfate may be used since it will tend to act in the same manner as sulfur in increasing soil acidity. However neither sulfur nor the acid fertilizers should be used indiscriminately but only after careful consideration of the needs of the soil and upon the advice of experts qualified to make recommendations concerning such matters.

Briefly, the control measures for the common scab of potatoes may

be summarized as follows: (a) Select the cleanest seed available. (b) Disinfect the seed by a standard method. (c) Rotate crops. A four or five year rotation is usually advisable. (d) Do not apply lime, ashes, manure or any alkaline substance to the soil just before planting potatoes. (e) Under competent advice apply sulfur or acid fertilizer to the soil before planting potatoes.

#### LABORATORY STUDY OF POTATO SCAB

**A. Symptoms.** — Examine potato tubers that show the symptoms of common scab. Are all the variations of symptoms which are described in the text found on the specimens at hand? Examine sections of scabby tissue under the microscope and note the formation of cork layers. Draw to illustrate all symptoms observed. Is any part of the plant other than the tuber ever attacked? With what other potato tuber diseases may common scab be confused? How may it be distinguished from each of these?

**B. The organism.** — The potato scab organism is so inconspicuous in the host tissue that it cannot well be studied except in artificial cultures. If cultures are available, note the gross appearance of the colonies then mount some of the fungus for microscopic examination. The chief features of the morphology of the organism are illustrated in Figure 177.

*Life history.* — Look up the salient features regarding the perpetuation and dissemination of this organism. What environmental conditions favor scab infection and development? What conditions inhibit scab development?

**C. Notes.** — Write full notes on this disease, being careful to distinguish it from other potato diseases, especially powdery scab and Rhizoctonia, and to discuss its relation to environmental factors.

#### REVIEW QUESTIONS

1. Describe the symptoms of common potato scab in detail so as to differentiate it from all other scab-like diseases.
2. Describe the pathological anatomy of the common scab lesions (See reference 14).
3. If absolutely scab-free seed potatoes are planted is a scab-free crop insured? Why?
4. Why is it not good policy to apply lime to the soil just before planting potatoes?
5. What other factors besides soil reaction are concerned in determining whether conditions are favorable or unfavorable for scab development?
6. Can potatoes be certified as scab-free by field inspection before harvest? Why?
7. How is sulfur used for scab control and in what does its value consist? What is inoculated sulfur? Is it better than common flowers of sulfur for scab control? Why?
8. Of what value is crop rotation in scab control? Seed treatment?

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## CHAPTER XXI

### DISEASES CAUSED BY ALGAE

In a treatise on plant diseases the Algae as a group are relatively unimportant. However, there are a few parasitic species which cause diseases of certain of the higher plants. Mann and Hutchison (5, 6) describe an alga, *Cephaleuros virescens*, which is parasitic on the leaves of *Camellia*, *Mangifera*, *Rhododendron*, *Thea*, *Croton*, and various ferns. In northern India and Assam this alga attacks the leaves and young shoots of the tea plant, *Thea sinensis*, causing a serious disease which is termed "Red Rust." This disease is most destructive as a stem parasite. In 1893 Lagerheim (4) reported a parasitic red alga, *Rhodochytrium spilanthis*, on a species of *Spilanthes* from Ecuador. In 1908 Atkinson (1) found this same species of *Rhodochytrium* on ragweed, *Ambrosia artemisiacifolia*, from South Carolina. More recently the question has been raised as to whether *Rhodochytrium* can be classified properly as an alga since it has no chlorophyll. Many species of red algae which are parasitic on other algae have been described (7, 8, 9) but these are of little interest to the plant pathologist.

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## CHAPTER XXII

### DISEASES CAUSED BY PARASITIC SEED PLANTS

Among the seed plants there are many species which are partially or wholly parasitic upon other species of seed plants. In the great majority of cases the damage done is so slight or the host plant is of such little economic importance that the matter attracts little attention except on the part of a few specialists who happen to be interested in the subject more from the standpoint of pure science than from any great practical bearing which such parasitism may have. However, there are a few instances in which parasitic spermatophytes attain considerable economic importance because of the damage resulting from their attacks upon valuable crop plants. There are several families of spermatophytes which contain species which are more or less parasitic. Among these are the mistletoe family (Loranthaceae), the morning glory family (Convolvulaceae, or the more restricted Cuscutaceae of some taxonomists, to which the dodders belong), the broomrape family (Orobanchaceae), the sandalwood family (Santalaceae) to which the bastard toad flax (*Commandra* spp.) belongs, and the figwort family (Scrophulariaceae) represented by the Indian paint brush or painted cup (*Castilleja*). Some of the species included in these various families are parasitic on the roots of the host plant, while others grow attached to the stem of the supporting plant. Some are devoid of chlorophyll and entirely dependent upon the host plant, while others have more or less chlorophyll and are only partly dependent on other plants for foods or raw materials. Not all species in all of the families mentioned above are parasitic. In fact a majority of the species in some of the families, as the figwort family and the morning glory family, are entirely independent, only one genus in each family containing parasitic species. The different types and degrees of parasitism occurring among these species of seed plants may be indicated in the following outline:

#### A. Stem parasites.

1. Entirely dependent; e.g., Dodder.
2. Partially dependent; e.g., Mistletoe.

#### B. Root parasites.

1. Entirely dependent; e.g., Broomrape (*Orobanche*).
2. Partially dependent; e.g., Bastard toad flax (*Commandra*).



The mistletoes and dodders are probably the most important economically of any of the parasitic phanerogams. They will be discussed more at length in the following pages. The broomrapes have also been known to do some damage as parasites on the roots of hemp and tobacco (2, 3), but instances of such damage are by no means as common as in case of dodder and mistletoe. The partial parasitism of species of *Commandra* (1) on the roots of various plants, including cherry and apple trees, has been observed but there is no evidence that any serious injury results from such attacks. The Indian paint brush, *Castilleja* spp., is a partial parasite on the roots of non-cultivated plants but is apparently of no economic importance. Thus from the standpoint of plant pathology we are probably justified in spending only a brief time in the study of this group of parasites but on account of the importance of certain species such as the mistletoes and dodders this phase of the subject warrants brief attention.

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#### Mistletoe Diseases

Caused by species of *Phoradendron* and *Razoumofskyia*

The mistletoe family includes a large number of species occurring in various parts of the world. The family exhibits various degrees of parasitism ranging all the way from complete independence as illustrated by a single species occurring in Australia, through various degrees of partial parasitism, to the total parasitism exhibited by a South American species growing upon a cactus. The European and North American species are all partial parasites which attack many different species of trees, both hardwoods and conifers.

The name "mistletoe" at once suggests the traditional sentiment which for centuries has been associated with this plant in Europe where it is always sought after as a Christmas decoration. This tradition has been brought to the United States and is now firmly fixed, especially in sections where the mistletoe is available. It is true that the American species are not identical with the European forms, but one of the species

in the eastern United States so closely resembles the common European species that the early settlers supposed it to be the same plant with which they were familiar at home and of course immediately invested it with the same traditions which surround the European mistletoe. Thus the great majority of people think of the mistletoe in terms of this traditional sentiment and do not at all realize that it is a pest which causes great damage to trees in some sections of the country.

**Distribution and Hosts.** — The mistletoes occurring in the United States belong in two genera of the family, Loranthaceae, namely, *Phoradendron* and *Razoumofskya*. There are several species of the latter genus, known as the dwarf mistletoes, all of which attack conifers. On the other hand the numerous species of *Phoradendron*, with few exceptions, are found on the non-coniferous trees. The species of *Phoradendron* are distributed throughout the southern United States, extending from the south Atlantic seaboard westward across the continent. They range as far north as New Jersey, southern Pennsylvania, southern Ohio, Indiana, Illinois, Missouri and eastern Oklahoma. On the Pacific Coast one or two species range northward through Oregon and Washington. The species of *Razoumofskya*, on the other hand, are confined largely to the western part of the country, occurring on conifers from the Rocky Mountains to the Pacific Coast. Most of the species found in the United States, together with their hosts and specific distribution, are given in the following lists.

Among the species attacking conifers are:

*Razoumofskya campylopoda* (Engelm.) Piper. On yellow pine. Northwestern United States.

*R. cryptopoda* (Engelm.) Coville. On yellow pine. Central and southern Rocky Mountains.

*R. americana* (Nutt.) Kuntze. On lodge pole pine. Western United States.

*R. cyanocarpa* (A. Nelson) Rydberg. On five-needle soft pines in western United States.

*R. divaricata* (Engelm.) Coville. On pinon pines in central and southern Rocky Mountains.

*R. laricis* Piper. On larch in northwestern United States.

*R. douglasii* (Engelm.) Kuntze. On Douglas fir (*Pseudotsuga*). Pacific Coast and Rocky Mountains.

*R. tsugensis* Rosend. On hemlock in northwestern United States.

*R. abietina* (Engelm.) Abrams. On fir (*Abies*). Rocky Mountains and western United States.

*R. pusillum* Pk. On *Picea* and *Larix*. Newfoundland and eastern Quebec to Pennsylvania and northern Michigan.

Species of *Phoradendron* found on non-coniferous trees include:

*Phoradendron cockerellii* Trelease. On ash, poplar and willow. Texas and New Mexico.

*P. coryae* Trelease. On oak. Arizona and New Mexico.

*P. eatoni* Trelease. On ash. Florida.

*P. engelmanni* Trelease. On alder, hackberry, oak. Texas.

*P. flavescens* Nuttall. On apple, ash, beech, birch, chestnut, cherry, elm, hackberry, hickory, linden, locust, maple, oak, plum, poplar, sycamore, walnut, willow. In central and southeastern states.

*P. longispicum* Trelease. On alder, ash, locust, oak, poplar, sycamore, walnut, willow. California and Arizona.

*P. macrophyllum* Cockerell. On alder, apple, ash, cherry, hackberry, locust, plum, poplar, sycamore, walnut and willow. In Arizona.

*P. macrotomum* Trelease. On ash, cherry, gum (*Nyssa*), oak, plum. In Florida.

*P. villosum* Nuttall. On locust, oak, poplar, willow. Pacific Coast.

A few species of *Phoradendron* are known to occur on conifers, among which are:

*P. libocedri* Howell. On Incense cedar (*Libocedrus*). Oregon, California and Nevada.

*P. juniperinum* Engelmann. On juniper. Colorado, Utah, New Mexico.

*P. ligatum* Trelease. On juniper. Pacific Coast and Nevada.

*P. densum* Torrey. On juniper. Pacific Coast.

*P. capitellatum* Torrey. On juniper. New Mexico and Arizona.

*P. pauciflorum* Torrey. On cypress. California and Arizona.

*P. bolleanum* Eichler. On juniper. Texas.

**Symptoms.** — The most evident indication of mistletoe infection, of course, is the presence of the mistletoe plant itself on the branches of the host (Fig. 178). This is especially true of the large leafy forms of *Phorodendron*. On badly infested deciduous trees the large bunches of mistletoe are very conspicuous during the winter season while the leaves are off the tree. Some of the dwarf mistletoes, *Razoumofskyia* spp., are very small and inconspicuous (Fig. 179) and therefore not so easily detected as the large leafy species. The first noticeable effect upon the host after a mistletoe plant becomes established upon it is usually, though not always, a swelling at the point of attack. Fusiform swellings are common on both conifers and hardwoods. The branch beyond the point of infestation usually ceases to grow and in many cases eventually dies so that the clump of mistletoe terminates the branch. Large branches and even trunks may be greatly deformed where infection occurred while the tree was young. On conifers large burls are

often formed, and practically all mistletoes on conifers cause witches' broom effects sooner or later. In some cases the brooms are rather stiff as on the yellow pine, while in other cases, as on Douglas fir, the brooms may be pendulous or of the "weeping-willow" type. Not all brooms on Douglas fir, however, are pendulous.



FIG. 178. — Mistletoe (*Phoradendron villosum*) on a branch of an oak tree.

**Economic importance.** — In view of the current popular knowledge of mistletoes it is no doubt surprising to the great majority of people to learn that the mistletoes are of considerable economic importance in that they seriously injure trees in some sections of the country. It is probably true that in Europe and in the southeastern part of the United States the damage caused by these parasites is negligible, but in the western United States there is evidence that mistletoes cause serious injury to trees. Bray (1) states that in certain semi-arid regions of

Texas, for instance, species of *Phorodendron* threaten the very life of the rather scant tree population of that region. Trees are at a premium in such regions because, due to climatic conditions, the struggle for existence is strenuous. Under such conditions trees seem to be more susceptible to mistletoe injury. Weir (11), and Korstian and Long (4) have described the mistletoe injury to coniferous forest trees in the Rocky Mountains and westward and have shown that the loss of timber from this source is great. The losses are of three general types: (a) actual death of trees, especially the younger ones; (b) reduction in the

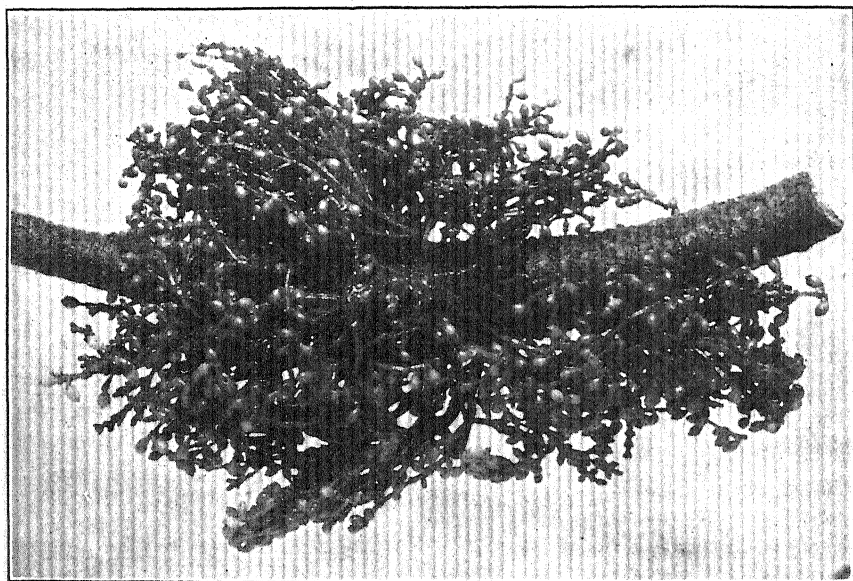


FIG. 179. — Dwarf mistletoe (*Razoumofskyia americana*) on lodge-pole pine.

annual increment due to the devitalizing effect of the parasite; and (c) secondary infestation by fungi and insects following mistletoe injury. Young seedlings, especially, are often killed by the parasite. Older trees sometimes succumb after a long period of heavy infestation. In some cases the upper part of a tree may be killed resulting in the stag-head or spiketop condition. Even when trees are not killed outright the reduction in amount of healthy foliage results in a greatly lessened annual increment. Large burls on the trunk and branches also interfere with the life processes and cut down the growth. The burls on trunks and large branches afford easy entrance for wood-rotting fungi and insects. Branches which are badly broomed often break off from the

heavy weight, especially when covered with snow. The branch stubs thus formed also provide a means of access by which wood-rotting fungi reach the heart-wood of the tree. The conifers which are most subject to injury by the dwarf mistletoes in the western United States are the larch (*Larix occidentalis*), lodge pole pine (*Pinus contorta*), Douglas fir (*Pseudotsuga taxifolia*), and yellow pine (*Pinus ponderosa*). In the central and southern Rocky Mountain region the mistletoe causes the most damage in the stands of western yellow pine.

**Life cycle of mistletoe.**— All the mistletoes are seed plants and consequently new plants start from seeds. The seeds are borne in berries and are surrounded by a sticky gelatinous matrix which enables them to cling tenaciously to the bark of trees. Birds eat the berries and are probably responsible to a certain extent for dissemination. In some species the seeds are forcibly ejected from the berries at maturity and stick to the bark of branches wherever they happen to lodge. On germination the hypocotyl forms a disk-like appressorium which flattens out against the host. From the center of this disk a root-like "sinker" penetrates the tissues of the twig and establishes connection with the water-carrying tissues (Fig. 180). After the mistletoe plant is established, cortical roots radiate in the bark of the host and send additional sinkers into the wood. A cross section of a branch at the base of the mistletoe parasite will show these wedge-shaped "sinkers" or parasitic roots penetrating the branch in a radial direction (Fig. 181).

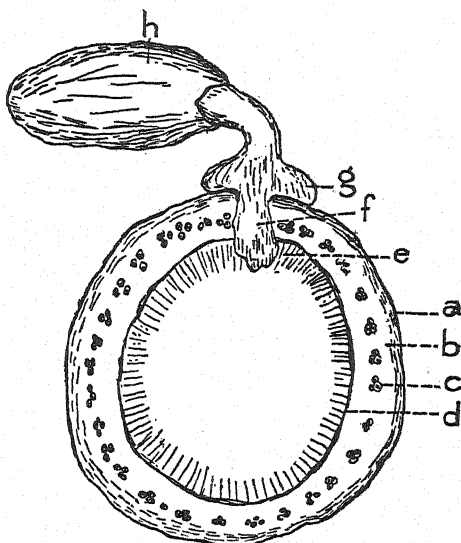


FIG. 180. — Germinating seed of mistletoe showing the manner in which the seedling becomes established on the host tree. a, corky covering of branch; b, cortex zone; c, bast fibers; d, cambium ring; e, wood zone; f, sinker of mistletoe penetrating the wood zone; g, disk; h, body of mistletoe seed. (After Bray, U. S. Dept. Agr., Bur. Pl. Ind. Bul. 166.)

There is some question as to the exact symbiotic relationship existing between the different species of mistletoe and their hosts. The large leafed species of *Phoradendron* apparently have abundant photosyn-

thetic powers and perhaps depend on the host only for water and the raw mineral elements coming from the soil. The dwarf mistletoes have little or no leaf surface and they are sometimes paler in color than the larger forms. It would seem that they are perhaps more dependent upon the host than some of the species of *Phoradendron*. However, Weir (11) performed an experiment which seems to throw doubt on the exact relationship existing here. He removed all leaves from six young

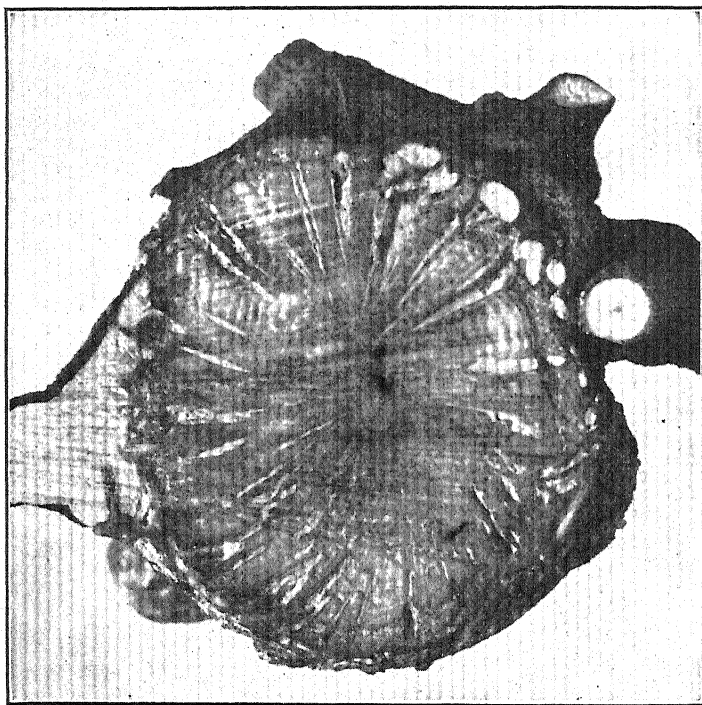


FIG. 181. — Cross section of an oak branch showing the parasitic nature of mistletoe. The wood of mistletoe is lighter in color than oak wood. Note the wedge-shaped parasitic roots of the mistletoe penetrating the host along the medullary rays.

lodge pole pines on which large clumps of mistletoe were growing. Six healthy non-infested trees of similar age were also defoliated. At the end of two years the defoliated trees containing no mistletoe were dead while those treated likewise but infested with large bunches of mistletoe were still alive. Did the mistletoe manufacture food for the tree? This is an interesting question worthy of further investigation. The fact remains, however, that all evidence points toward the conclusion that mistletoes are harmful to trees in a great many cases.



**Control.** — In Texas, where street trees and other scattered trees are infected, Bray (1) suggests that these may be treated by breaking or scraping off the mistletoe plants and by pruning off small infested branches. In the coniferous forests of the West the problem of control is quite different. It is closely related to the method of forest management and logging operations. Mistletoe dies when its host tree is cut. The general policy in logging a tract infested with mistletoe should be to leave no infested trees standing because the mistletoe develops rapidly on scattered trees left on a cut over tract since these trees are exposed to more light which favors mistletoe growth. The seed trees left standing should be free from infestation in so far as practicable.

#### LABORATORY STUDY OF MISTLETOE

**A. Symptoms.**— Examine branches of trees infested with mistletoe plants of different ages. Note any enlargements at the point where the parasite is attached to the host. How does the size of the branch on the distal side of the mistletoe compare with its size on the side next the tree? Examine the bark over the surface of the swollen area for evidences of the roots of the parasite partially imbedded in the bark. Observe cross sections of the host branch cut at the base of the mistletoe plant. Can you distinguish the tissues of the parasite from those of the host? Describe the appearance of the cross sectional view. Are the symptoms caused by species of *Phoradendron* similar to those caused by species of *Razoumofskya*? Draw to illustrate the symptoms. Which genus attacks mostly the coniferous trees and which the non-conifers?

**B. Life history of mistletoe.** — How do the leaves and stems of *Phoradendron* differ from those of *Razoumofskya* in size and color? Is chlorophyll present in both genera? Which seems to have the most intensive chlorophyll coloration? Describe the reproductive organs of mistletoe. Do the flowers and fruits of the two genera differ in any way? Describe the manner of seed dissemination. Describe the processes of seed germination and infection. How do the root tissues of the mistletoe plant make contact with the tissues of the host so as to facilitate absorption? What elements are absorbed from the host? What part of the food supply of the mistletoe plant does it make for itself?

**C. Notes.** — Write notes describing the different species of mistletoe examined, the symptoms caused by them, the life cycle of a mistletoe, and the nature and extent of the damage caused by these parasites.

#### REVIEW QUESTIONS

1. Discuss the parasitic relationship existing between mistletoes and their hosts.
2. Describe the life history of a mistletoe.
3. In what part of the United States are mistletoes of economic importance?
4. What are the two genera of mistletoes occurring in the United States? In general what type of trees serve as hosts for the species of each genus? Are there any exceptions?
5. Under what conditions do mistletoes seem to cause serious injury to trees?
6. In connection with which coniferous trees does mistletoe injury occur most extensively?
7. Discuss control measures in the coniferous forest.



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## Dodder

Species of *Cuscuta*

Dodder is usually considered by the grower as an ordinary weed, yet it is a parasite in the true sense of the word. It draws its elaborated foods as well as water directly from its host plant whereas ordinary weeds compete with crop plants for water and the other raw food materials from the soil. Species of dodder are widely disseminated over the world on a wide range of hosts, yet on the whole there are comparatively few crops that are seriously attacked by this parasite. Dodder is most troublesome on certain leguminous crops as clover and alfalfa and on flax. Species have been seen occasionally on various vegetables such as onions and tomatoes; and dodders are not uncommon on many wild herbaceous and shrubby plants, as, for example, willow, golden-rod, ragweed, hazel, smartweed, etc. The fact that dodder seeds are of such size as to readily contaminate seed stocks of

clover, alfalfa and flax, and the fact that dodder seeds mature at about the time the seed crop of clover, alfalfa, or flax is ready to harvest are undoubtedly important factors in making dodder a more serious pest of these crops than of any others.

A large number of species of dodder are found in America but only a comparatively few of them are of any great economic importance. A few of the more important species are listed below.

1. Clover dodder (*Cuscuta epithymum* Murr.). — This species was introduced into this country from Europe. It occurs in parts of the West and is found also in the northeastern United States. It prefers clover and alfalfa but is not confined entirely to these hosts.

2. Small-seeded alfalfa dodder (*C. planiflora* Ten.). — This dodder is not limited to alfalfa but is particularly troublesome on this crop. In the United States it occurs only in the West, having been introduced from Europe.

3. Large-seeded alfalfa dodder (*C. indecora* Choisy). — This is a native species, preferring alfalfa but sometimes occurring on other plants. It occurs mostly in the West, rarely in the East or South.

4. Field dodder (*C. arvensis* Beyrich). — This is a native species which is of most importance east of the Mississippi River but is widely distributed over the country. It is not partial to any particular crop, having a wide range of hosts.

5. Flax dodder (*C. epilinum* Weihe). — This species was introduced from Europe and is found in the northeastern United States and Canada.

6. Common dodder (*C. gronovii* Willd.). — Another native. Widely distributed in the United States as far west as Montana and Texas. Attacks a wide range of hosts.

7. Chilean dodder (*C. racemosa chiliana* Engelm.). — Introduced from South America where it is common on clover and alfalfa. Not feared in this country.

**The dodder plant.** — The dodder plant is a slender twining vine with minute, rudimentary, scale-like leaves, and almost or entirely devoid of chlorophyll in all its parts (Fig. 182). The color of the plant is usually yellow. In some cases, plants take on an orange color and some species have a reddish or purplish tinge. The flowers are very small and occur in dense clusters. The plants are annuals, reproducing by seeds which ripen in late summer or autumn. The seeds germinate on the ground in spring and the young seedling is able to lead an independent existence only as long as the stored food in the seed is available. If the young dodder vine does not come in contact with a congenial host plant about which it can twine, it soon perishes. In case it does find a host it soon establishes a parasitic relationship and continues growth until seeds are



FIG. 182. — Clover plants attacked by dodder. Note the vine twining closely about the clover stem and the abundance of blossoms and seed pods. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept., 1911-12.)

matured (Fig. 183). The entwining vine puts forth parasitic roots in the form of suckers or haustoria which penetrate the tissues of the host

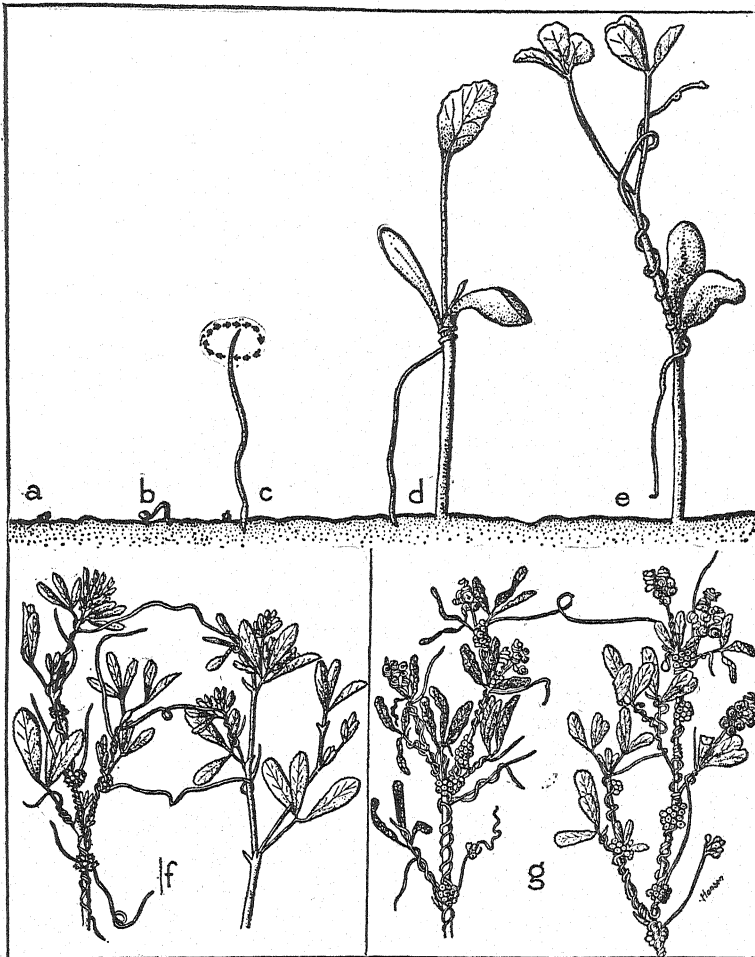


FIG. 183. — Germinating seeds and seedlings of dodder. The seedling can live independently until the food stored in the seed is exhausted. If it succeeds in fastening upon a host plant before the reserve food is used up, as at d, it severs its connection with the ground as at e, and lives entirely upon the host plant. If no host is available the dodder seedling soon perishes. (After Hansen, U. S. Dept. Agr., Farmers' Bul. 1161.)

and connect with both the water-carrying channels and the food-carrying elements of the host stem (Fig. 184). As soon as contact is thus established with the host the root and lower portion of the dodder

vine perish, thus severing all connection with the soil and henceforth the parasite grows entirely upon the host plant and is wholly dependent upon it for sustenance. The vegetative stems of the dodder plant spread extensively from plant to plant in a dense stand of clover or alfalfa, matting together and smothering down large patches of the forage plants.

**Control.** — As a rule no special control measures are necessary for this pest except in case of such crops as clover and alfalfa where the dodder seeds are carried as impurities in the seed of these crops. Extensive infestation of fields by the clover and alfalfa dodders is not apt

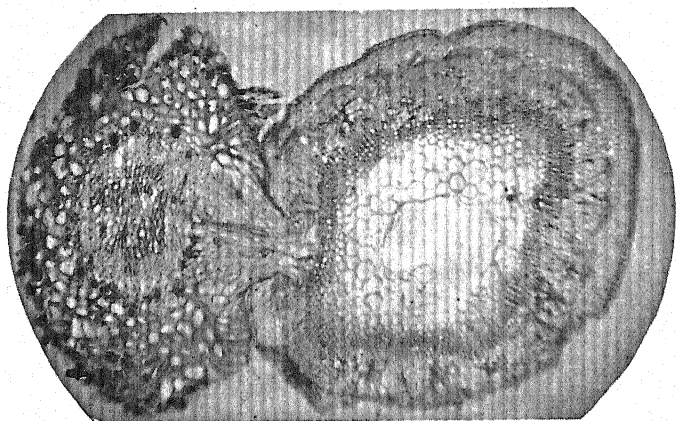


FIG. 184. — Cross section of alfalfa stem attacked by dodder. Alfalfa at right, dodder at left. Note how the parasitic root (haustorium) of the dodder penetrates the alfalfa stem. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept., 1911-12.)

to occur if absolutely clean seed is used. The removal of dodder seed from impure seed is a difficult task and requires special equipment. This is especially true in case of the large-seeded alfalfa dodder, the seeds of which are almost the same size as clover and alfalfa seed. Other species of dodder have smaller seeds which can be screened out by the use of sieves with meshes of the proper size. Where seed is too badly contaminated it is better to secure seed known to be free from dodder seed.

If dodder is discovered in a field the infested patches should be mowed before the dodder seed has matured. When the cut plants are dry they should be burned. In case dodder has matured seed and contaminated a whole field it may be necessary to plow it up and plant to crops which are not injured by this parasite. Dodder seeds will live in the ground for four or five years. Early mowing and close pasturing subsequently may eventually eradicate the weed without plowing up the field.

## LABORATORY STUDY OF DODDER

**A. Symptoms.** — Examine specimens of crop plants such as clover, alfalfa, flax or any other plant infested with dodder. What effect has the dodder plant upon the plant which it attacks? In what manner is the damage done? If possible, observe the dodder in the field where it has infested considerable areas of clover or some other crop. Note the smothering effect. Is the smothering out of crop plants the only item of economic importance in connection with dodder?

Examine samples of clover or alfalfa seed which contain an intermixture of dodder seed. Learn to distinguish the dodder seeds from crop seeds of similar size and appearance. Can the dodder seeds be separated readily from clover seed? Of how much importance, commercially, is such adulteration of seed stocks?

**B. Life history of the dodder plant.** — Note the structure and color of all parts of the plant. Has it any leaves? Any chlorophyll? Examine the reproductive parts. Are they typical of the seed plants? Under what conditions will the seeds germinate? Where does the seedling begin its existence? How long can it lead an independent life? How does it become attached to the host plant?

Examine a cross section of the stems of both dodder and host cut at the point where a haustorium penetrates the host plant. Identify the different vascular elements of both plants and note the contact made between corresponding elements in host and parasite. What food elements does the dodder absorb from its host? Can the dodder plant manufacture any part of its food supply? Why?

**C. Notes.** — In the notes discuss the economic importance of dodder, the crops most often attacked, the appearance of the dodder plant, its life history and methods of control.

## REVIEW QUESTIONS

1. Describe the life history of a dodder plant from seed to seed. Where does the seedling start its growth? Where does it get its nourishment at first? Where does the mature dodder plant get its sustenance?
2. What is the nature and extent of the injury done by dodder?
3. How does dodder differ from mistletoe in the matter of nutrition? In nature and extent of injury caused?
4. Discuss dodder as a seed adulterant.
5. Discuss remedial measures, both in the field and in seed stocks.

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## CHAPTER XXIII

### DISEASES CAUSED BY NEMATODES

The nematodes or eelworms constitute a very large group of animal life belonging to the zoölogical group commonly referred to as round-worms. The round-worms are widely distributed over the earth's surface and infest a wide range of hosts. Many of them parasitize other animals. The species of nematodes which are of particular interest to the plant pathologist are soil-infesting organisms which spend a part of their life cycle in or on living plants causing disease symptoms of various kinds. The plant-inhabiting eelworms are very minute organisms, scarcely visible to the unaided eye, except the greatly enlarged female of certain species which can readily be seen with the naked eye. Some of the more important plant diseases caused by nematodes include: 1. The root-knot caused by *Heterodera radiculicola*; 2. The nematode disease of sugar-beets caused by *Heterodera schachtii*; 3. The wheat nematode disease caused by *Tylenchus tritici*; and 4. The leaf and stem disease caused by *Tylenchus dipsaci*.

#### Root-knot

Caused by *Heterodera radiculicola* (Greef) Müller

This nematode disease in plants has been known for over seventy years in Europe and for a somewhat shorter period in the United States. Various names have been applied to the malady, among which may be mentioned root-knot, root-gall, club-root, big-root, eelworm disease and nematode disease. Berkeley (3) mentioned this disease in England as early as 1855. It was reported from Germany in 1864 as a root-knot disease on certain grasses. Later reports have indicated the existence of the trouble in other countries of Europe, in various countries of Asia and the East Indies, in Africa, in South America and the West Indies, and in Australia and New Zealand. In the United States the root-knot disease was reported as a greenhouse pest in 1876 (13). In this country, as a widespread pest in the field, it first attracted attention in some of the southern states. At present it is widely disseminated, especially in the southern part of the United States, but is sometimes found in



many of the northern states, even being reported from as far north as Michigan, though it seems to be more at home in tropical and sub-tropical countries. The original home of this eelworm is not definitely known but it seems logical to suppose that it is indigenous to the tropics and that it has been introduced into Europe and temperate America on some of its native hosts which have been transplanted to these regions.

**Hosts.** — The list of susceptible species of plants reported by various writers in the several countries where the pest has been studied totals over 500 different kinds of plants. These include species belonging in many different families and represent all kinds of cultivated and non-cultivated plants, both herbaceous and woody. Some of the more important cultivated plants which are subject to the root-knot disease are named in the following list: alfalfa, asparagus, almond, bean, beet, begonia, cantaloupe, carrot, catalpa, celery, cherry, clematis, clover, coleus, cotton, cowpea, cucumber, dahlia, eggplant, European grape, field pea, fig, flax, garden beet, garden pea, ginseng, hollyhock, lettuce, mulberry, muskmelon, onion, okra, peach, pecan, peony, pepper, Persian walnut, potato, pumpkin, rose, salsify, soy bean, spinach, strawberry, sugar beet, sugar cane, sweet pea, sweet potato, tobacco, tomato, vetch, violet, watermelon.

While it is true that the majority of crop plants are susceptible to attack by nematodes, there is still a considerable number of important cultivated plants which are immune or only slightly susceptible. The immune or resistant crops include barley, corn, certain varieties of cowpea (Brabham, Iron, Monetta, Victor), many grasses, kafir, milo, peanut, millet, redtop, rye, sorghum, a variety of soy bean (Laredo), timothy, velvet bean, wheat, and winter oats.

**Economic importance.** — The eelworm causing root-knot is apt to infest greenhouses in any part of the country, and while it is not possible to give accurate estimates of the losses involved they are undoubtedly considerable. In the field most of the serious losses have been confined to the southern part of the United States. In some of the southern states cotton and many of the truck crops are frequently damaged seriously. In some sections of South Carolina the loss of cotton has been estimated at over 4 per cent of the crop. In 1917 the loss to the cotton crop in Georgia was estimated (10) at 4 per cent of the crop, and for the entire South it is estimated that root-knot causes an average annual loss of 200,000 bales of cotton and 100,000 tons of seed. In Florida the losses in such truck crops as snap beans, cabbage, celery, eggplant, potatoes, lettuce, peas and tomatoes have been estimated at from 1 to 25 per cent in the various crops. The disease is also serious in parts of the South-

west. In parts of southern California root-knot is making serious inroads on the melon crops. Such nursery stock as figs, grapes, peaches and walnuts also suffers from eelworm infestation.

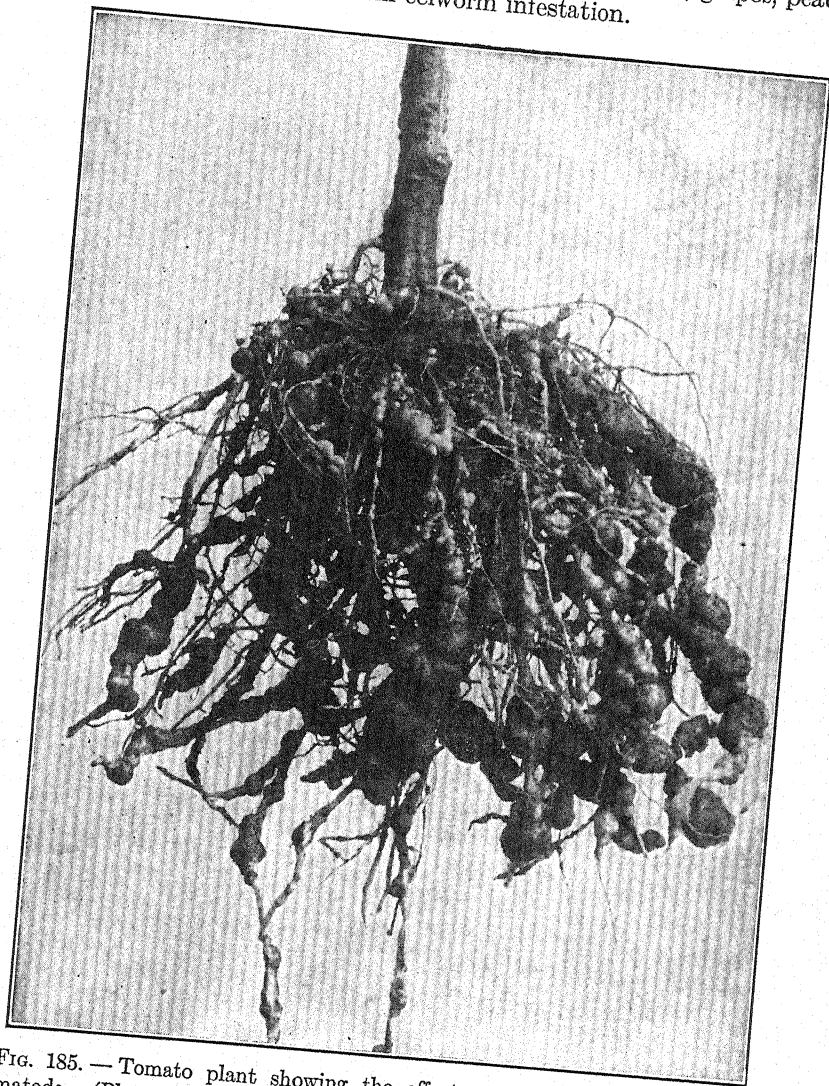


FIG. 185.—Tomato plant showing the effects of invasion by the root-knot nematode. (Photograph by McKay, Ore. Agr. Exp. Sta.)

**Symptoms.**—Two sets of symptoms should be considered in describing the effect of the disease on the host plant, namely, the root symptoms and the effects as exhibited by the parts of the plant above

ground. As the name indicates, the characteristic symptoms on the roots consist of knots or swellings of various shapes and sizes. The enlargements may occur at intervals on the root giving a beaded effect

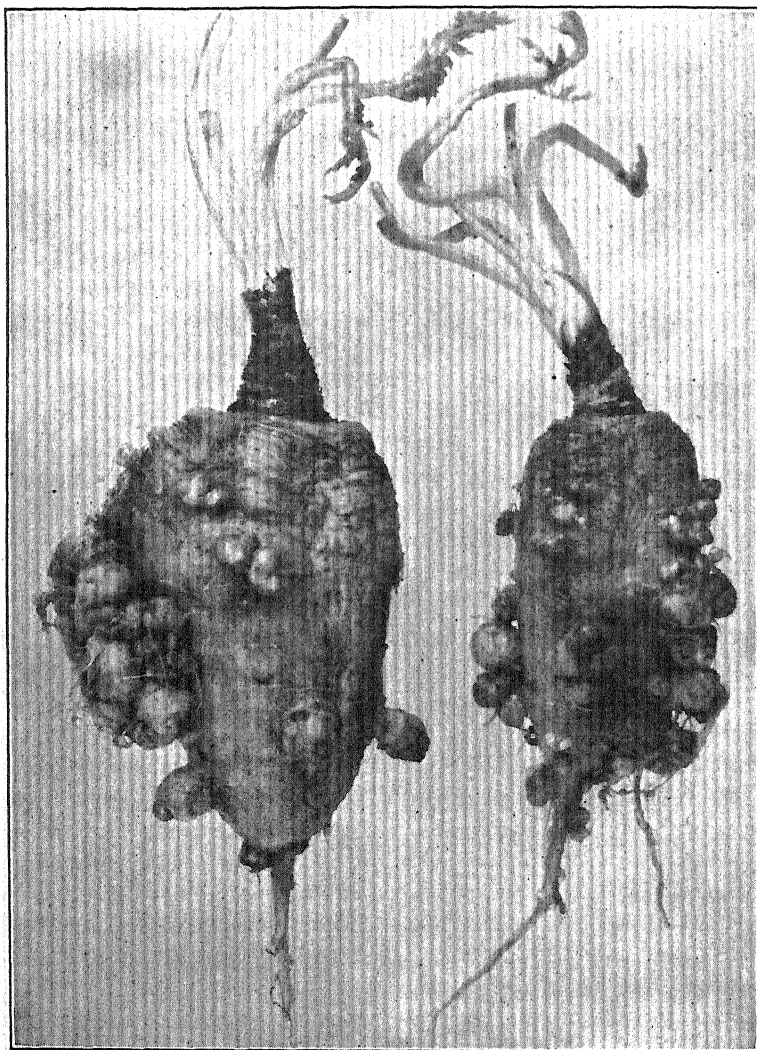


FIG. 186. — Root-knot on carrots. (Photograph by McKay, Ore. Agr. Exp. Sta.)

or the root may be more or less swollen throughout its length. Only a part of the roots may be attacked or the whole root system of a plant may be affected. The knots vary in size depending somewhat on the size of the infected root. Figure 185 represents a tomato plant with a

severe case of root knot. Nematode root-knots should not be confused with the nodules produced by the nitrogen-fixing bacteria on plants of the legume type. The latter are small spherical or lobed bodies attached to the side of the rootlets and may be easily detached, while the root-knot eelworms cause swellings in the roots themselves.

This particular nematode does not cause malformations or enlargements on the above-ground parts of the host plant. Nevertheless plants with badly infested roots present a more or less sickly appearance of the aerial parts. They are dwarfed and wilt more readily in hot dry weather than plants with healthy roots. The foliage is usually a paler green than that of healthy plants. Such symptoms above ground may be due to other causes, however, therefore to make the diagnosis certain the roots should be examined for the characteristic symptoms. A still further test is the demonstration of the presence of the eelworms in the tissues of the knots. These can usually be found by teasing out a bit of the knot tissue in water and examining it with a microscope.

**Structure and life cycle of the parasite.** — The female nematode lays numerous eggs from which the larvae are hatched. The eggs are ellipsoidal to kidney-shaped bodies averaging about 85 to 98 by 34 to 40  $\mu$  (4). The larva which emerges from an egg is a slender cylindrical organism measuring 375 to 500  $\mu$  in length by 12 to 15  $\mu$  in greatest diameter. The anterior end is blunt and the posterior end tapers to a pointed tail. The structure of this larval animal may be simply described as consisting of two telescoping tubes; the alimentary canal within the body wall. The former consists of a chitinous spear, 10 to 15  $\mu$  long, at the anterior end which may be considered as a part of the mouth apparatus, since it can be used in boring through cell walls and in taking nourishment. Back of the spear extends the esophagus which expands into the esophageal bulb. Behind the bulb the alimentary canal is narrow but soon expands into the large digestive tract which fills the body cavity. The larvae are able to remain alive in the soil for several months under favorable conditions. If they are able to enter the roots of a host plant they continue to develop into the adult nematodes, otherwise they eventually die. Within a suitable host the larvae grow to maturity. Up to this time the sexes are not distinguishable.

The adult individuals of *Heterodera radiculicola* show a marked difference between the male and the female. The male is a long slender worm, resembling the larval stage in most respects but with some differences. It is greatly increased in size, measuring 1200 to 1500  $\mu$  in length and 30 to 36  $\mu$  in diameter. The tail is short and more rounded than in the larval stage. The spear is somewhat larger than in the larva. Compare the male and female of *Tylenchus dipsaci* (Fig. 191).

It is in the adult female, however, that the greatest differentiation occurs. She is somewhat flask-shaped or pear-shaped (Fig. 187), measuring 400 to 1300  $\mu$  in length by 270 to 500  $\mu$  in greatest diameter. The anterior end is not greatly enlarged but just posterior to the esophageal bulb the body abruptly enlarges so that the whole posterior portion of the adult female is greatly swollen and rounded into an almost spherical shape. Each female is capable of laying 500 or more eggs.

*Overwintering.* — The fact that the larvae can remain alive in the soil for several months probably accounts for a great deal of overwintering, although in case of perennials, or volunteer plants of annual crops which survive the winter in the milder climates, the nematodes undoubtedly survive in the host tissues either as larvae or adult females. The overwintering is comparatively easy in those countries where the winters are mild and little or no freezing occurs. According to Bessey (4), however, these parasites are able to survive the severe winters of our northern states also.

*Dissemination.* — The nematode larvae are capable of slight and slow locomotion but at best they are probably able to travel by their own efforts only a very few feet during a season. They are disseminated very largely by man on propagating stock of various kinds. Shipments of nursery stock, tubers, bulbs and seedlings from infested areas are most likely to blame for the widespread dispersal of the root-knot nematode. Infested soil may be carried from field to field on implements and vehicles. Overflow water may carry infested soil to a clean field.

*Infection.* — Infection usually takes place in the younger roots although this nematode is able to bore its way into older roots and has even been known to penetrate stems and fruits of plants such as melons and tomatoes when these come in contact with the soil. Potato tubers are also frequently invaded. The presence of the parasite in the host stimulates the tissues in its vicinity to excessive cell division and growth thus resulting in the hypertrophies so characteristic of this malady.

*Favorable environment.* — The most favorable soil for the propagation of the root-knot nematode is a light sandy one. The animal will not flourish in a heavy compact soil. Lands that remain very wet or water-soaked for long periods during the year are not apt to harbor many of these eelworms. A warm temperature is also necessary for the best development of the organism. In soils with a temperature below 55° F.

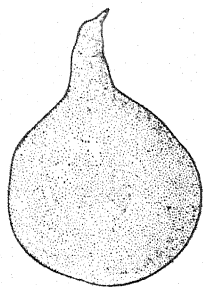


FIG. 187. — The female of *Heterodera radicicola*. The males and the larvae are similar in general appearance to those of *Tylenchus dipsaci*. See Fig. 191.

the worms do not thrive and there is little danger of infection. Severe freezing kills nematodes unless they are deep enough in the ground to be protected. Repeated applications of manure or humus in other form to a heavy soil may render it more favorable for nematode infestation. The soils that are brought in and built up for greenhouse use usually offer a very favorable environment for nematodes. Some of the worst infestations occur in greenhouses. The warm light sandy soils occurring over much of the southern United States are very suitable for the development of these pests and consequently the heaviest losses from this trouble occur in the southern states.

**Control.** — Manifestly the control of this pest centers around the eradication of the organisms from the soil. There are two means of accomplishing this end, namely, (a) sterilization of the soil, and (b) crop rotation. In the greenhouse and in seed beds soil sterilization is feasible and is practiced extensively. Two general sterilizing agents are available, chemicals and heat. Poisonous chemicals such as formaldehyde have been tried as soil disinfectants for eelworms but have not proved as satisfactory as steam for this purpose. Where steam is available and can be applied through perforated pipes laid in the soil it has given satisfactory results when used for this purpose. (For various methods of applying steam see Chapter VIII.) Sometimes it is advisable to replace the soil in greenhouse benches. New soil should be secured which is known to be free from nematodes, but before placing it in the greenhouse the benches should be thoroughly disinfected to kill any worms left behind when the old soil is removed. For large areas in the field the only feasible means of eradication once the ground has become infested is crop rotation. Some of the resistant crops mentioned above under host plants should be used in the rotation. The susceptible crops must be kept off the land for at least two years and no susceptible weeds should be allowed to grow among the resistant crop plants used in the rotation. Practically all of the nematode larvae will perish in less than two years if they have no susceptible plants to live upon.

**Prevention.** — Where a grower has land free from this pest he should take every precaution to prevent its introduction on nursery stock or any other propagating stock which he brings in from other localities. In case a few infested trees or other stock are planted they should be dug out and destroyed as soon as discovered. The soil occupied by the infested roots should then be sterilized to prevent the spread of the disease to other parts of the field.



## LABORATORY STUDY

**A. Symptoms.** — Examine specimens of any available plant which shows the results of nematode attacks. Note the swellings of various shapes and sizes. Do they occur on the main root or on lateral rootlets? Can you notice any of the above-ground symptoms on these plants? Make habit sketches to illustrate all symptoms observed.

**B. The organism.** — Dissect bits of diseased tissue by teasing it out in water. Look for the worms with a microscope. Try to distinguish larvae as well as male and female adults. Sometimes one can find the females by splitting or tearing the tissue apart rather than by cutting. The females may be seen with the naked eye partly imbedded in the torn surface of the specimen. They appear as tiny white or pearly beads easily visible to the unaided eye. Carefully remove one and examine with a magnifier or the low power of a microscope. The males resemble the larvae except that they are much larger. Look for eggs. Draw all stages found.

**C. Life history.** — Look up all available information on the life history of this eelworm and fix the facts in mind so that you can discuss control measures as based on these facts.

**D. Notes.** — Write a complete account of this disease and its causal organism together with a discussion of control measures.

## REVIEW QUESTIONS

1. Name a list of crop plants which are susceptible to root-knot.
2. Name a list of resistant crops.
3. What is the nature of the organism causing this trouble? Describe its life history.
4. How does this organism differ from the other large groups of organisms causing plant diseases?
5. What environmental conditions are favorable for the development of root-knot? Unfavorable?
6. What life-history facts form the basis for control practices?
7. On what life-history fact is the recommendation for soil sterilization based? What is the best sterilizer for this purpose?
8. Under what conditions is crop rotation rather than soil sterilization to be recommended?

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### The Sugar-beet Nematode Disease

Caused by *Heterodera schachtii* Schmidt

The sugar-beet eelworm was first reported from Germany where a large sugar industry had grown up during the first half of the nineteenth century. By the middle of that century there was a notable falling off in the yield in the oldest beet-growing districts. Later it was found that this reduction in yield was due to heavy nematode infestation. In 1908 the pest was discovered in California and is now well established in some of the beet-growing districts of California and Utah. The sugar-beet nematode is closely related to the common root-knot nematode and its morphology is very similar. There is one striking difference in its life history, however. The female of *H. schachtii* dies while still filled with eggs and becomes a brown sac-like cyst in which the eggs may lie dormant for many years, a few hatching each year and the larvae escaping from the cyst. Experimental work has shown that in badly infested soil many encysted eggs can still be found five years after beets were last grown on the land. However it was shown that the nematode population decreased so greatly after a four or five year rotation that one good crop could be grown on the land, but if a second consecutive crop were planted the yield dropped off again due to the increase of nematodes on the first crop.



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FIG. 188. — Strawberry plants infested with the leaf and stem nematode, *Tylenchus dipsaci*. (After McKay, Ore. Agr. Exp. Sta. Crop Pest Rept., 1915-20. 1921.)

### Stem, Leaf and Bulb Nematode Disease

Caused by *Tylenchus dipsaci* (Kuhn) Bastian

This disease affects chiefly the stems and leaves of plants rather than the roots. This statement holds in spite of the fact that this nematode causes serious trouble in certain bulb crops, because bulbs consist essentially of modified underground leaves. The nematode causing this disease occurs in various parts of Europe, Australia, New Zealand,



FIG. 189. — Alfalfa plant showing effects of invasion by *Tylenchus dipsaci*. (Photograph by McKay, Ore. Agr. Exp. Sta.)

and South Africa. The disease has been known since 1851 and the early literature concerning it is entirely European. The malady seems to have been known in the United States for about twenty years. In this country it is known chiefly in the far western states, although it has been reported from certain states east of the Rocky Mountains. The nematode, *Tylenchus dipsaci*, has been found on over fifty different

hosts including narcissus, hyacinth, onion, garlic, strawberry, clover, alfalfa, lupine, broad bean, kidney bean, wheat, oats, barley, rye, potato, hops, flax, turnip, plantain, buttercup, dandelion, false dandelion, sow thistle and daisy. In the United States the more important cultivated crops which have been found severely infested with this nematode are narcissus, clover, alfalfa and strawberry. In recent years the disease has been found quite abundantly on all these hosts in certain localities in the Pacific Coast states. There is strong evidence that many biological strains of this eelworm exist. In Oregon there is good reason to believe that the strain on the false dandelion, *Hypochoeris radicata*, is different from that on the strawberry. The population on the bulb crops also evidently belongs to a different strain from that on the strawberry and on *Hypochoeris*.

**Symptoms.** — On such plants as strawberry, clover, alfalfa and false dandelion the symptoms consist of swellings or galls on any part of the

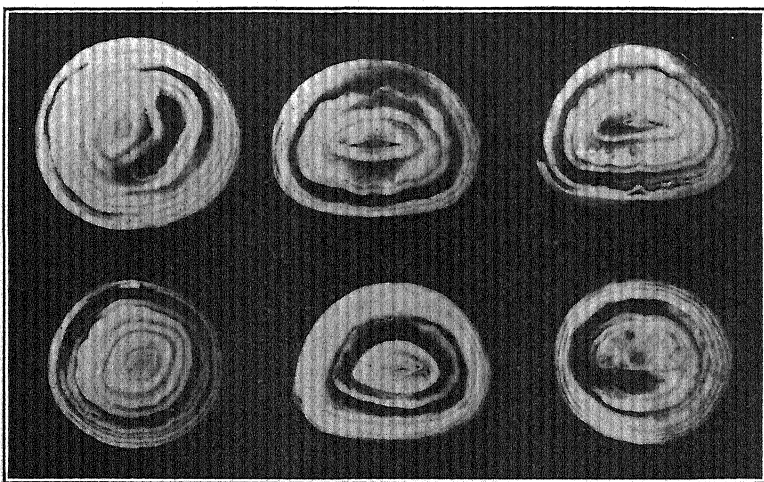


FIG. 190. — Nematode disease of narcissus bulbs. Cross sections of the diseased bulbs show some of the layers darkened due to invasion by the nematodes. (After McKay, 18th Ann. Rept. Ore. Hort. Soc., 1926.)

leaves and stems (Fig. 188). The leaves become crinkled, deformed or dwarfed. Affected plants are apt to appear stunted or dwarfed in comparison with healthy plants. In bulbous crops dark rings appear in a cross section of the bulb where the worms have migrated from certain leaves into the fleshy leaf-bases in the bulbs. This condition gives rise to the term "ring disease" (Fig. 190). The affected leaves of such crops show yellow streaks or specks rather than extreme swellings or

malformations such as occur on the strawberry, although diseased narcissus leaves may be somewhat twisted or distorted and exhibit characteristic thickened specks which can be felt beneath the epidermis rather than on the surface. The life history of the eelworm causing this trouble is illustrated in Figure 191.

**Control.** — The control of this disease in bulbs consists of roguing out diseased plants that can be detected in the field, and in treating bulbs used for propagation. A hot water method has been devised which has proved successful. It consists of immersing the bulbs in water held at 110–111° F. for 3 hours. This is sufficient to kill the worms in the bulbs without injuring the bulbs. In case of diseased strawberries all diseased plants should be rogued out and destroyed, or if the planting is badly infested it should be plowed up entirely and the land planted to non-susceptible crops for two or three years. Care should be exercised in setting out new plantings to see that the sets come from clean stock.

#### LABORATORY STUDY OF LEAF AND STEM NEMATODE

**A. Symptoms.** — Examine diseased specimens of strawberry, clover, alfalfa, and narcissus bulbs. Note the characteristic swellings of leaves and stems. Observe the characteristic symptoms in cross sections of the bulbs. If weed hosts such as plantain and false dandelion are available compare effects on these plants with the specimens of cultivated hosts. Break open the swollen parts and note the texture of the hypertrophied tissue. Make drawings to illustrate all symptoms.

**B. The nematode.** — Tease out bits of diseased tissue from any of the affected plants in a few drops of water and examine with a microscope. Find the worms swimming free and identify the different stages, larvae as well as male and female adults. Compare with the same stages of

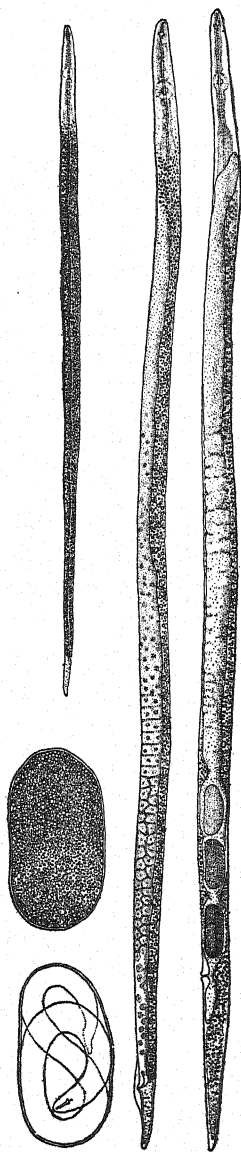


FIG. 191. — *Tylenchus dipsaci*. Right, adult female. Center, adult male. Upper left, larval stage. Center left, egg, very highly magnified. Lower left, egg containing an embryo eelworm, very highly magnified.

the root-knot eelworm and any other species studied. Make drawings of all stages found.

C. Notes. — Write notes comparing the leaf and stem nematode with others studied.

#### REVIEW QUESTIONS

1. Compare the leaf, stem and bulb nematode with the root-knot nema and the sugar-beet nema as to the morphology and life cycle of the causal organism.
2. Compare the symptoms of this disease with the root-knot disease.
3. Compare the host range of the leaf, stem and bulb infesting nematode with that of the root-knot nematode.
4. What ornamental bulb plants are particularly susceptible to this nema?
5. What successful means of treating infested bulbs has been devised?

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### The Nematode Disease of Wheat

Caused by *Tylenchus tritici* (Steinbuch) Bastian

This is a disease of wheat which manifests itself in the above-ground parts of the plant, being most noticeable as an affection of the grains. It is known in many parts of the world but is not as generally distributed as is the root-knot disease. The first definite report (9) of this disease indicates that it was present in England in 1743. It was reported from continental Europe (4) in 1775, and again in 1799 Steinbuch (10)

discussed a nematode disease of wheat and referred to the causal organism as *Vibrio tritici*. In 1864 Bastian (1) transferred the organism to the genus *Tylenchus*, retaining the specific name, *tritici*, as used by Steinbuch. A great many papers have appeared in Europe on this disease but it was not until 1909 that the trouble was reported in the United States. Johnson recorded it from California in that year and also stated that it had been found in New York, West Virginia and Georgia. In 1917 Fromme (7) reported the occurrence of the wheat nematode in Virginia. At the present time the wheat nematode is known to occur in all of the continents except Africa. It has been found in France, Germany, Austria, Switzerland, Italy, Sweden, Holland, England, Australia, China, Brazil, and in North America. There is also evidence that it occurs in Russia, Turkestan and India (4). In the United States it has been definitely reported from at least seven states, namely, New York, Virginia, West Virginia, Georgia, North Carolina, South Carolina and California.

**Economic importance.** — Some of the early accounts of this disease in Europe indicate that it caused severe losses. More recent reports do not record any extensive damage resulting from its attacks. There does not seem to be any very definite information as to the reduction in yield of wheat due to this disease in the various other foreign countries where it is known to occur, but such evidence as there is indicates that the disease may be quite severe in some instances. In the United States the most extensive damage yet reported occurred in Virginia. Losses of from 1 to 50 per cent of the crop have been noted in some wheat fields in that state.

**Hosts.** — The eelworm, *Tylenchus tritici*, confines its attacks largely if not entirely to wheat. There are nematodes which attack many other species of grasses but there is a difference of opinion as to their specific rank. Some are inclined to the opinion that the forms found on other species of grasses are simply strains of the wheat nematode. On the other hand, there is good reason for holding that these forms are of specific rank and some investigators have given them specific names, using the name, *T. tritici*, to designate the form which attacks wheat only. The difference seems to be physiological, however, rather than morphological, since attempts to inoculate other grasses with the wheat nematode have failed, except in case of emmer, rye and spelt on which inoculations with *T. tritici* produced abundant infections.

**Symptoms.** — The wheat nematode infests only the aerial parts of the host. The symptoms appear on the leaves and in the head where the ovaries are attacked. Infected leaves exhibit a wrinkling, twisting or rolling, especially of the younger leaf blades (Fig. 192). On the

upper surface of the leaf small, elevated, rounded areas may appear. The leaves may become yellow, wilt and die. The diseased spikes are easily detected. They are usually reduced in size and the glumes spread outward giving the head a somewhat thickened and shortened appear-

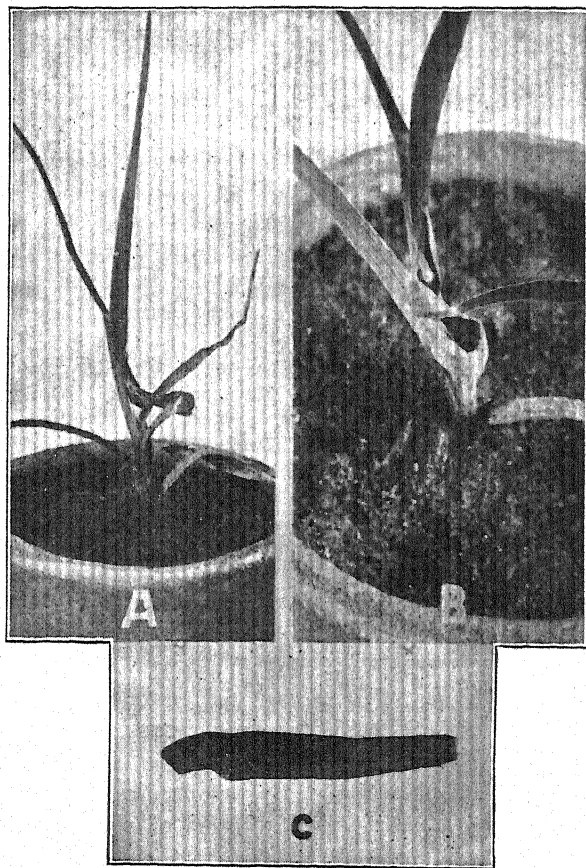


FIG. 192. — Young wheat plant showing effects of infestation by the wheat nematode. A, five-weeks-old wheat seedling grown in the greenhouse and artificially infected with *Tylenchus tritici*. B, part of the plant shown in A enlarged. C, leaf blade of a wheat seedling showing the one-sided apical development and small nematode galls resulting from artificial inoculation in the greenhouse. (After Byars, U. S. Dept. Agr. Bul. 842.)

ance. Depending upon the extent of the infestation a varying number of the florets contain galls instead of sound kernels of wheat. These galls are hard, light brown to dark-colored, shorter and usually thicker than normal grains and are filled with nematodes. The spreading of



the glumes and also the color of the galls give this disease a striking resemblance to the bunt or stinking smut, but the smut balls are lighter in weight and more easily crushed than nematode galls. Figure 193

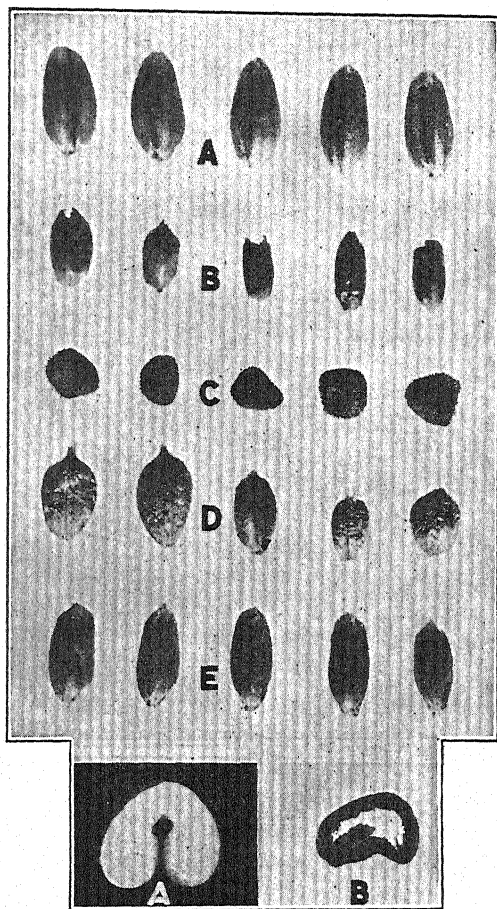


FIG. 193. — Comparison of nematode infested wheat kernels with normal grains and other objects with which they may be confused. Above: A, normal grains; B, nematode galls; C, seeds of cockle, a weed sometimes found in wheat fields; D, smutted wheat grains (smut balls); E, bin-burnt grains. Below: A, section of normal wheat grain; B, section of grain infested with nematodes. (After Byars, U. S. Dept. Agr. Bul. 842.)

shows the difference in the general appearance of smutted grains and those infested with nematodes.

**Life history.** — The general life cycle of this eelworm resembles very closely that of the root-knot nematode. A female of this species may



lay more than 2000 eggs. The larvae emerge and go through a period of growth before differentiating into male and female. In this species the female does not become pear-shaped as is the case with *Heterodera radicola* but remains cylindrical in shape although becoming somewhat more thickened in diameter than the male.

The larvae apparently may overwinter either in the galls which fall to the ground or are sown with fall wheat, or on the seedlings which they may invade in the fall. They locate between the leaf sheaths

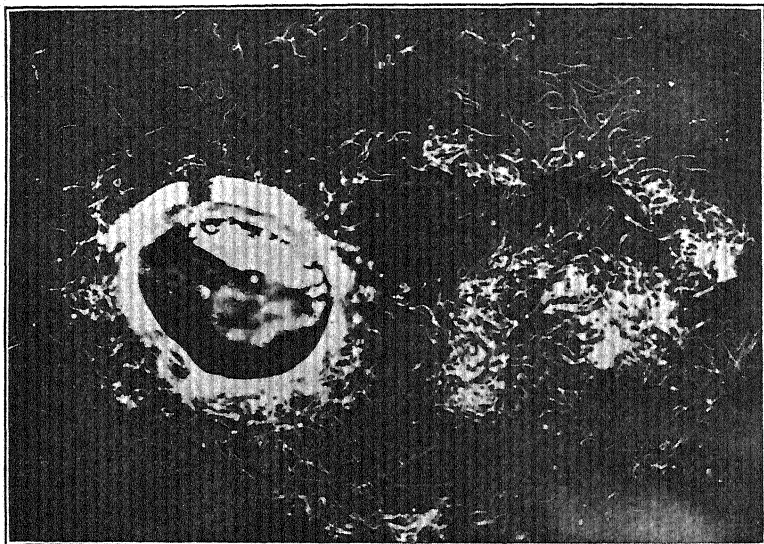


FIG. 194. — Larvae of *Tylenchus tritici*. Mass of thread-like eelworms escaping from a cut-open flower gall.  $\times 6$ . (After Byars, U. S. Dept. Agr. Bul. 842.)

near the terminal bud. Here they are in a position to attack the leaves and also the ovaries of the wheat head when it emerges. Inside the host tissues they develop into adult males and females. Within each kernel gall a cavity is formed in which the mature females lay thousands of eggs which soon hatch into larvae. As many as 90,000 larvae have been found within one large sized gall. Medium sized galls contain on an average about 15,000 larvae (4).

*Dissemination.* — In the dry galls the larvae may remain alive and dormant for years. It is in this condition that they are dispersed over long distances. Importations of wheat from infested districts afford the greatest chance for long distance dissemination. The use of seed from an infested field may spread the disease in the neighborhood. The larvae may live for several months when freed in the soil but probably

few survive for more than a year as free-living organisms in the soil. In the soil they are subject to the same kinds of transportation as the root-knot nema, such as drainage water, farm implements and vehicles.

**Control.** — The control of this disease is not as difficult as that of many other diseases. There are just two items of importance to consider: (a) clean seed, and (b) crop rotation. The best method of obtaining clean seed, of course, is to secure it from a source where no disease exists. If this is not possible, the nematode galls may be separated from seed wheat by pouring the wheat into a brine consisting of a 20 per cent solution of common salt. The nematode galls, light grains and smutted kernels will float and can be skimmed off. The sound grain should then be rinsed in clear water and dried before sowing. A crop rotation of 2 years or more will rid a soil of contamination. Since the host range of this nematode is so limited it is easy to find other crops for the rotation.

#### LABORATORY STUDY OF WHEAT NEMATODES

**A. Symptoms.** — Examine wheat plants and grains infested with *Tylenchus tritici*. Compare the foliage symptoms with those caused by *Tylenchus dipsaci*. Are the wheat stems affected in any way? Note the effect on wheat kernels. Compare with grains of wheat infected with stinking smut.

**B. The organism.** — Break open an infested grain and examine the contents with the microscope. Compare the worms found with other species studied. Look up the life history of this species. What part do the galled grains play in the cycle?

**C. Notes.** — Write notes comparing this disease in all details of symptoms and life history with other nematode diseases studied.

#### REVIEW QUESTIONS

1. Which of the nematode diseases discussed in preceding pages most resembles the wheat nematode disease?
2. Of which of the preceding diseases is the causal organism most closely related to the wheat eelworm?
3. Of the four eelworm diseases discussed in this text, which has the most limited host range?
4. How is the wheat eelworm disseminated to new localities?
5. With what other wheat troubles may the nematode-infested wheat grains be confused? How distinguished from these?
6. Discuss control measures for the wheat nema.

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## CHAPTER XXIV

### THE VIRUS DISEASES OF PLANTS

The question of the proper classification of that group of plant diseases which is now quite generally known as the virus diseases has been a puzzling one. For a long time two general classes of plant diseases have been recognized, namely, the parasitic diseases and the non-parasitic group. The former includes all that large group of diseases which

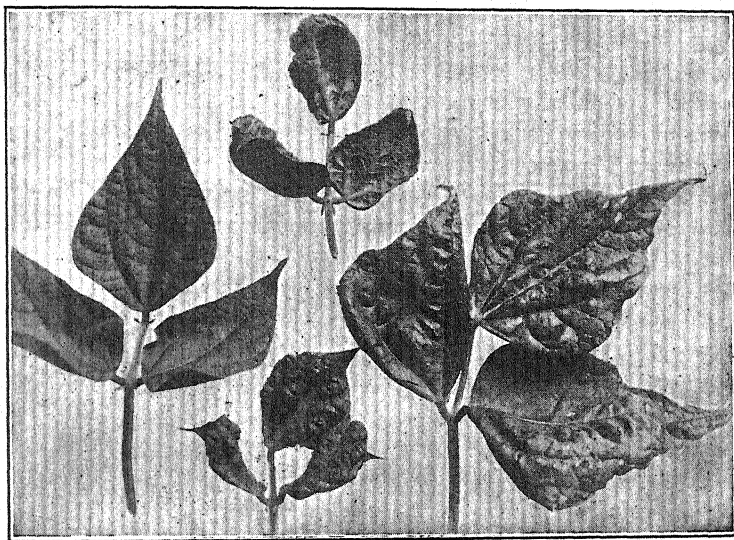


FIG. 195. — Bean leaves affected with mosaic. Healthy leaf at left. (After Barss, Ore. Agr. Exp. Sta. Third Crop Pest Rept., 1915-20. 1921.)

are definitely known to be due to parasitic organisms. To the latter class belong the diseases which without doubt are not due to the attack of parasites, but to other factors, largely, if not entirely, environmental. These non-parasitic diseases have frequently been referred to as "physiological" diseases because they were conceived to be due to a derangement of the normal physiological processes of the plant, which was not induced by any parasite. When cases of mosaic and other troubles of similar nature came to the attention of plant pathologists, there was naturally doubt as to the exact nature of these diseases. In the earlier investigations of these maladies there was a marked tendency to class

such disturbances as "physiological." This was quite natural since they could not be attributed to any definite cause, but seemed to partake more of the nature of other non-parasitic or "physiological" diseases which were better known. As knowledge of the infectious nature of these diseases increased, investigators began to look more and more for an organism as the causal agent. However, as yet it has not been positively proved to the satisfaction of all plant pathologists that these diseases are due to a parasitic organism, in spite of the fact that many observations point in that direction, and until this fact is definitely established this group of diseases cannot be classed with the well-known parasitic diseases. Until the facts are all known it seems best to put these diseases tentatively in a class by themselves. Because of the peculiar infectious nature of this type of disease in which the "contagium" or "virus" is carried in an unknown form in the plant juices, the term "virus diseases" or "viroses" has been quite generally adopted to designate the class.

**Symptoms.** — At this point we will discuss the symptoms of the virus diseases in a general way leaving the detailed description of particular

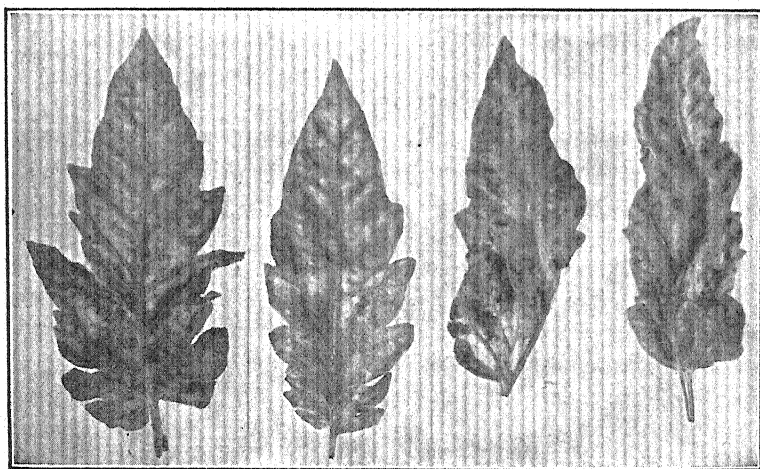


FIG. 196. — Tomato leaves showing mosaic symptoms. (After McKay, Ore. Agr. Exp. Sta. Third Crop Pest Rept., 1915-20. 1921.)

symptoms to be taken up later under the specific virus diseases of particular crops which have been selected for more extended treatment in succeeding pages. In general, the different types of symptoms attributable to viruses may be classified as (a) mottling, (b) chlorosis, (c) distortion, (d) dwarfing and attenuation, and (e) necrosis. The mottling of foliage was one of the symptoms which gave rise to the term

"mosaic" which is one of the first types discussed in the early literature of virus diseases. The symptoms of mosaic as originally defined consist of a mottled appearance of the foliage due to a difference in intensity of the green color, making a mosaic-like pattern of alternating dark-green and paler yellowish-green spots or blotches on the leaves. More recently modifications of these symptoms have been observed, giving rise to such terms as mild mosaic (Fig. 199), rugose mosaic (Fig. 201) and leaf-rolling mosaic, which are now recognized as three distinct virus diseases of the potato. Chlorosis is a common symptom among

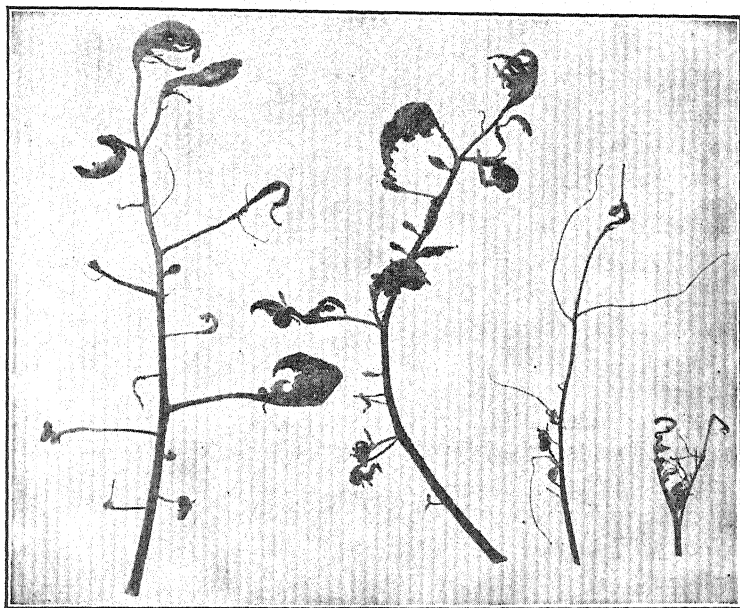


FIG. 197. — Tomato leaves showing extreme attenuation due to mosaic. (After McKay, Ore. Agr. Exp. Sta. Third Crop Pest Rept., 1915-20. 1921.)

these diseases. The lighter areas in ordinary mosaic are due to a chlorotic condition. In peach yellows and strawberry yellows, a general chlorosis is one of the manifestations of disease. Distortion is one of the very common symptoms of many of the virus troubles. It consists of puckering, cupping, curling or rolling of the leaves in various ways. Examples of distortion are found in rugose potato mosaic, bean mosaic (Fig. 195), tomato mosaic (Fig. 196) and cucumber mosaic (Fig. 204). In the latter the fruits as well as the leaves show striking deformities (Fig. 205). Dwarfing and attenuation or abnormal slenderness are strikingly shown in witches' broom of potato (Fig. 203) and in tomato

mosaic (Fig. 197). A necrotic condition develops in some of the virus troubles, notably in rugose mosaic, leaf-roll and witches' broom of potato. In some cases the necrotic lesions develop in the foliage while in certain of the potato virus diseases they occur in the tubers.

An interesting feature in connection with symptoms of the virus diseases is the "masking" of symptoms under certain conditions. In case of the foliage symptoms in certain potato virus disorders, such as rugose mosaic and mild mosaic, higher air temperatures inhibit the appearance of the symptoms. At temperatures of 88° F. or above it is difficult to detect such symptoms as mottling, rugosity, curling or rolling of the foliage. The spindle tuber of potato, on the other hand, is an example where high soil temperatures tend to bring out the symptoms rather than to inhibit them. This phase of the virus disease problem is one which has received limited attention, however, and no doubt future investigations will develop interesting facts in this connection.

**Hosts.** — Since the early work of Mayer (26) on tobacco mosaic, published in 1886, many different types of virus diseases have been identified and the list of host species known to be susceptible to one or more of them is constantly increasing in number. Members of the night-shade family (Solanaceae) are especially susceptible and a number of them were among the first on which these diseases were studied. No attempt to give a complete list of susceptible species here will be made, but a partial list is included to show the widespread prevalence of this class of plant diseases. Among the families known to contain susceptible genera and species are the following: Nightshade family (Solanaceae), Rose family (Rosaceae), Gourd family (Cucurbitaceae), Legume family (Leguminosae), Carrot family (Umbelliferae), Milkweed family (Asclepiadaceae), Buttercup family (Ranunculaceae), Mint family (Labiatae), Mallow family (Malvaceae), Unicorn-plant family (Martyniaceae), Spurge family (Euphorbiaceae), Goosefoot family (Chenopodiaceae), Pokeweed family (Phytolaccaceae), Composite family (Compositae) and Grass family (Gramineae). Some of the plants which are susceptible to one or more virus diseases are tobacco, potato, tomato, species of ground cherries (*Physalis*), horse-nettle (*Solanum carolinense*), Jamestown-weed (*Datura stramonium*), petunia, cucumber, pumpkin, raspberry, blackberry, strawberry, peach, sugar-cane, corn, spinach, celery, lettuce, turnip, cabbage, milkweed (*Asclepias syriaca*) poke-weed (*Phytolacca decandra*), spurge (*Euphorbia preslii*), dahlia, bean, and clover. Some of the viruses have been transmitted experimentally to many other plants. No one knows how many specific viruses there are, and much of the cross inoculation work attempted in the past is not



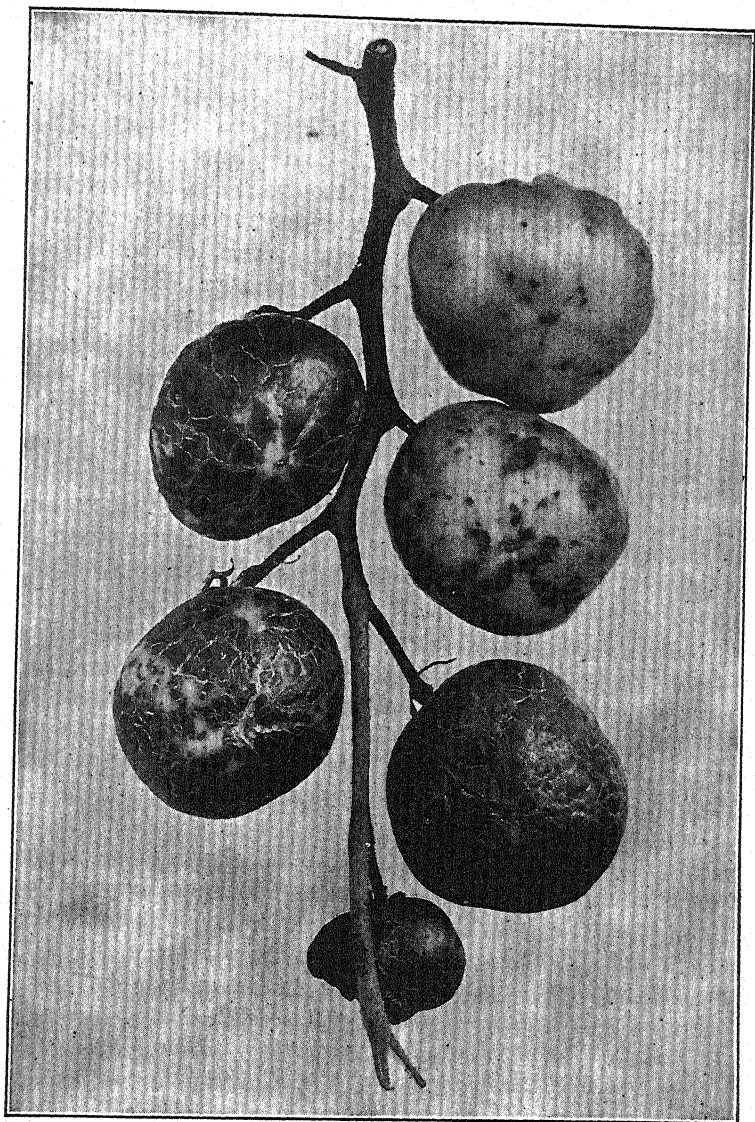


FIG. 198. — Mosaic symptoms on tomato fruits. (After McKay, Ore. Agr. Exp. Sta. Third Crop Pest Rept., 1915-20. 1921.)



conclusive because the identity of the virus in question has not always been definitely determined. This problem can probably be satisfactorily worked out only by the use of differential hosts (32) in much the same manner as in determining strains of wheat rust, for example.

**Cause.** — The problem of determining the exact cause of the virus diseases has been a baffling one. For many years opinion has been divided between two lines of thought. Some plant pathologists have been inclined toward the theory that the trouble is non-parasitic or "physiological" in nature, while others have maintained that the causal agent must be a parasite, even if they were, for the time being, unable to demonstrate the presence of the parasite. For the physiological theory two chief lines of argument have been advanced. One is based upon the belief that some sort of an enzyme is responsible, and the other is that the trouble may be due to excess or deficiency of nitrogen, carbohydrates or other elements necessary in the nutrition of the plant. The argument in favor of a parasitic origin of the trouble is based upon the fact that the juice of diseased plants carries an infective principle which will produce the disease in healthy plants when inoculated into them. This contagious nature of the virus diseases has now been demonstrated thousands of times. This has led many to believe that the cause is a bacterium of some kind. The inability to identify this supposed microorganism with the microscope and to culture it as other bacteria are cultured has led many to doubt this theory and to rely upon the "physiological" theory as the best explanation. More recently protozoa have been suggested as the cause of at least some of the virus diseases. These various theories will now be taken up more in detail and the main lines of argument of some of the exponents of each theory will be presented.

*The unbalanced nutrition theory.* — This theory was advanced tentatively by some of the early workers as a working hypothesis and considerable experimental work was done in an effort to determine whether or not this had any bearing upon the problem. The tobacco mosaic was one of the first virus diseases studied and much of the early as well as later experimental work has dealt with this particular virus disease. Mayer (26) in Europe was the first to study the tobacco mosaic carefully. His work indicated that the trouble was not due to insufficient mineral nutrients. His tests showed as much potassium salts, phosphates, calcium, nitrogen, and magnesium present in soils and plants where the disease was present as where it was absent. The trouble seemed to be distributed over a field regardless of soil conditions. He found that variations in moisture and temperature did not affect the occurrence of the disease. He finally proved that the juice of diseased

leaves injected into healthy plants would produce the disease. Woods (31) working with the same disease came to the conclusion that anything that causes a reduction in the available nitrogenous reserve food below that required to carry on active cell division may cause mosaic. Freiberg (17) carried out an extensive series of microchemical tests to determine if the amounts of different chemicals such as nitrogen, ammonia, iron, calcium, magnesium, potassium, phosphorus, and sulfur, as well as proteins and carbohydrates, varied in the light-green and the dark-green regions of diseased leaves. In the case of all the inorganic chemicals he found very little if any difference in the amounts present in the lighter and darker areas. He thought there might be slightly more total nitrogen in the paler areas but the difference was very slight. Of proteins there were apparently more in the light-green areas than in the dark-green areas. The carbohydrates were always in excess in the greener areas. After considering the results of these tests Freiberg came to the conclusion that the mosaic disease is not due to nutritional factors. In short as a result of these experiments and others the theory of a nutritional cause of the virus diseases has not been widely accepted.

*The enzyme theory.* — This theory concerning the causal agent of the virus diseases has been strenuously upheld by certain investigators. For some years the enzyme theory, as opposed to the bacterial theory, has held the center of the stage, each theory being championed by its own followers. More recently the protozoan theory has come more into the limelight. While Woods (31) believed that the trouble was due to a certain extent to lack of nitrogenous food reserves he thought that these nutritional disturbances were accompanied by an abnormal activity of oxidizing enzymes. This activity of the enzymes prevents the proper elaboration of reserve food. Woods went so far as to state that on the decay of the plants these enzymes are liberated into the soil where they remain active and may be absorbed through the roots of other plants. Freiberg (17) believed that an enzyme was responsible but disagreed with Woods as to the kind of enzyme concerned. He found no conclusive evidence that the enzymes concerned are oxidases but was inclined to believe that a special type of enzyme is responsible for the trouble. He was uncertain as to the nature of this enzyme, but suggested that it might be an aldehydase. He further concluded that this enzyme has the power of increasing when injected into healthy plants. Allard (5) proved conclusively that peroxidase and catalase can not be responsible for the mosaic disease, and as will be shown later, he thinks the disease is not due to an enzyme at all.

*The bacterial theory.* — Because of the infectious nature of the virus diseases and because they can be so easily transmitted from one plant

to another by the injection of juice from a diseased plant into a healthy one, many have been convinced that an organism of the bacterial type must be responsible for the trouble. The fact that no organism of this type has been seen or cultured has not deterred some pathologists from postulating an ultramicroscopic organism of some kind as the cause. Allard (5) has been among the foremost in advocating such a theory. In 1916 he asserted his belief in this theory in the following words: "Since this pathogenic agent is highly infectious and is capable of increasing indefinitely within susceptible plants, there is every reason to believe that it is an ultramicroscopic parasite of some kind."

*The "virus" theory.* — It probably is not correct to call this a definite theory as distinct from all other theories, but rather an attempt to cover up our ignorance, a subterfuge, as it were, for us to hide behind until our efforts to find the real cause are successful. The term "virus" is borrowed from human and animal pathology. It has been used by animal pathologists for a long time to designate a contagium that can not be definitely associated with a known organism. Some fifty "virus" diseases are now known in the animal kingdom. In general, attempts to culture these animal viruses have failed although certain investigators claim to have done so in a few cases. The infective principle seems to be too small to be seen by a microscope and in general not to be culturable by any of the methods known to bacteriology. Most of the animal viruses pass through standard porcelain and earthenware filters. In some of the animal virus diseases, cell inclusions which can be seen with the microscope are associated with the disease, but it is not definitely known whether these are the cause or the result of the disease.

When the infectious nature of the plant diseases of this type became known through the discovery that some kind of an infective principle is carried in the juices of diseased plants, the first impulse of course was to look for bacteria as the causal agent. But when persistent effort failed to find bacteria, either with the microscope or by bacteriological culture methods, the natural tendency was to note the striking resemblance between this type of plant disease and the virus diseases in animals and to adopt the term "virus diseases of plants" to designate this type of malady in the plant kingdom. The uncertainty as to the exact nature of the agency causing the trouble, coupled with a knowledge of certain characteristics of the agency gained through experimentation, has led to the use of various terms which have crept into the literature of the virus diseases. The terminology applied to the causal agency, whatever it is, includes such terms as "contagium vivum fluidum," "filterable virus," "contagium," "filterable contagium," "infective principle," and "causal agency." In the present

state of our knowledge these terms serve very well and some such terminology will probably continue in use until the problem is completely solved and the exact nature of the contagium is determined.

*The "gene" theory.* — In 1923 Duggar and Armstrong (12) attempted to formulate some sort of a concept as to the nature of the infective principle in this type of disease. They had previously determined (11) by a process of filtration that the infective particles, whatever their nature may be, are approximately 30-millionths of a millimeter in diameter. Their tentative conclusion is that the causal agency may be a product of the host cell, not a simple enzyme, but some structure with a definite heredity, possibly a particle of chromatin or a gene. They conceive of this "gene" as having gone wild or revolted from its usual course of action, and, being able to reproduce itself indefinitely in living cells, it causes abnormal disturbance in any cells into which it is introduced.

*Protozoa.* — During recent years some investigators have turned their attention toward cytological studies of plant cells affected with virus troubles. In these studies various peculiar bodies have been found as cell inclusions. In 1921 Kunkel (21) described certain bodies which he found in the cells of corn affected with mosaic. These bodies showed a reticulate structure, were vacuolate and resembled protoplasm in staining properties. They were always closely associated with the nucleus of the host cell. He invariably found these bodies in the cells of the light-green areas of the leaf and never in the dark-green areas. Kunkel made no definite claims as to what these bodies were, but suggested that they might be similar to those associated with certain virus diseases in man and animals. In 1923 McKinney, Eckerson and Webb (24) described somewhat similar bodies associated with wheat rosette and leaf-mottling. They were certain that the bodies they saw were not artifacts, but ventured no positive statements as to their exact nature. They might possibly be organisms of some kind or possibly the result of some reaction of the host cell itself toward the disease. They also stated that these cell inclusions resemble in a general way those peculiar bodies of unknown nature which are associated with certain of the virus diseases of animals, especially the bodies found in connection with rabies and smallpox. In making some cytological studies on bean, clover, tomato and potato plants affected with virus diseases, Nelson (28) described and figured flagellate bodies which he called protozoa. He was the first to state that an organism of protozoan nature is probably the cause of these so-called virus diseases. This work of Nelson's stimulated research along this line to a remarkable extent and many articles soon appeared taking issue with Nelson on this

subject. For a long time flagellates have been known in the latex of certain plants where they apparently are not associated with any mosaic-like symptoms. While much has been said adverse to the protozoan origin of these diseases, some workers, persistently pursuing these cytological studies, continue to find flagellate bodies associated with the diseases of this nature. In 1926 Eckerson (15) published the results of similar studies on tomato mosaic. She found numerous very small, motile, flagellate organisms in all diseased leaves. In many cases these organisms were found inside the chloroplasts. Her inoculation experiments were very interesting. Alternate leaflets were inoculated and at intervals of 24 hours the uninoculated leaflets opposite the inoculated leaflets were examined. After 24 hours the tiny flagellates were found in uninoculated leaflets opposite inoculated leaflets, but not in other uninoculated leaflets. Within three days after inoculation tiny flagellates and larger forms occurred in large numbers throughout the mesophyll and in five days many cells of the mesophyll were in bad condition. Chloroplasts were floating at random and many of them were completely liquefied. After ten days the leaflets began to show mottling.

These studies on the protozoan-like bodies found associated with the virus diseases are very interesting, but not conclusive. Many problems arise in attempting to ascribe the cause of this type of disease to such organisms. Among the questions arising is that of the life-history cycle in connection with the known properties of the contagium. It is difficult to understand how an organism of the protozoan type can undergo all the filtration and other tests to which the infectious juice has been subjected and still perpetuate the disease. No doubt these researches will continue, however, until the relation of protozoa to this type of disease is definitely determined.

**Properties of the virus.** — While doubt still exists as to the exact identity of the causal agency in the virus diseases of plants, yet a great deal is known concerning various characteristics of this infective principle. It has been definitely established in many cases that the contagium is in the sap of the plant and can be extracted by grinding up the diseased plant parts and expressing the juice. The extract thus secured can be used as an inoculum and the disease produced in healthy plants just as cultures of bacteria are used in producing the well known bacterial diseases of plants. It has also been found that certain insects, particularly sucking insects, can readily transmit the disease if they migrate to a healthy plant and feed upon it after feeding upon a diseased plant. In some cases, as in beet curly top, the disease has never been transmitted by artificial juice inoculations but is carried only by a

certain species of insect. This fact suggests that the causal agent is not identical in all cases of virus disease since the characteristics exhibited by the virus differ in some respects in the different cases of virus diseases.

When a small quantity of the infective principle is introduced into a healthy plant the virus is able to multiply or increase in amount. In this respect it resembles living organisms. It is readily transferable from the point of inoculation through the sap channels to other parts of the plant and, as a rule, after an incubation period of several days to a few weeks the virus is found in all parts of the plant. The virus hibernates in perennial living parts of plants. Specific instances of this are found in the potato viruses which are carried over in the tubers; in the tomato mosaic disease which is known to overwinter in the underground perennial parts of certain weeds, the horse nettle, *Solanum carolinense*, and the ground cherry, *Physalis* spp.; and in the cucurbit mosaic which hibernates in the milkweed, *Asclepias syriaca*, and others. There is a possibility that it also hibernates in certain insects. This point needs further investigation. The possibility of hibernation in the soil has also received attention but this question is in a state of uncertainty at present.

Expressed plant juices containing the virus have been subjected to a wide variety of tests in order to determine some of the properties of the infective principle. Allard (5, 8) has been one of the foremost in investigations of this kind. The effects of many different chemicals have been tried. In general it requires rather strong solutions of the various chemicals to kill the virus. Nitric and hydrochloric acids do not render the contagium inactive in concentrations of less than 1 gram in 50 to 100 cc. It requires a concentration stronger than 1 gram of potassium permanganate or zinc chloride in 100 cc. of water to kill the virus. Alcohol stronger than 55 per cent is highly toxic. Eighty per cent alcohol kills the virus in less than one-half hour. Four per cent formaldehyde is very toxic to the infective principle. Temperatures near the boiling point quickly kill the contagium. The virus is not so sensitive to low temperatures, being able to withstand a temperature of  $-180^{\circ}$  C. without losing its infectious properties.

After the infective nature of these diseases was discovered attempts were made to culture an organism by ordinary bacteriological methods. All these attempts failed. Finally Olitsky (29) claimed to have succeeded, by special culture methods which he devised, in multiplying the causal agent outside the living plant. He used a specially prepared extract of healthy tomato plants as a culture medium. It had previously been shown by Allard (4) and by Doolittle (13) that there is a limit to

which a plant juice extract containing the virus can be diluted and still retain its infective capacity. They both agreed that a dilution weaker than 1 : 10,000 loses the capacity to produce the disease when used as an inoculum on healthy plants. Olitsky claimed that by his cultural method he could use much more dilute solutions and still get infection. From this he reasoned that the infective agency must have multiplied in his cultures. Shortly after Olitsky's announcement, Mulvania (27) and Purdy (30) duplicated his experiments but were unable to verify his statements; all their results being negative. Up to the present time, therefore, there is no conclusive and undisputed evidence that the infective principle of these diseases can be multiplied or increased by artificial culture methods.

*Size of the infective particles.* — Quite early in the investigations of this type of disease it was shown that the infective principle would pass through filters which would retain ordinary bacteria. Later it was found that the contagium would not pass through a certain type of filter. Allard (5) found that the causal agent of tobacco mosaic held back when the juice is filtered through a Livingston atmometer porous cup. Doolittle (13) found that the contagium is not removed from the juice by filtration through a Berkefeld filter, but that juice from diseased plants is rendered non-infectious by passing through Chamberland filters. With these facts in mind Duggar and Karrer (11) attempted to determine more accurately the size of the infective particles. By using filters which would permit the passage of the particles, and others through which the particles would not pass, they were finally able to find a filter with pores just large enough to permit the passage of the particles. In this way they finally concluded that these particles are approximately 30-thousandths of a micron, or 30-millionths of a millimeter in diameter, and that the ratio 30 : 1000 roughly expresses the diameter relations of the virus disease particles in comparison with the average size of bacterial organisms causing plant diseases.

*Virus from apparently healthy potatoes.* — A very interesting phenomenon has recently been reported which has a bearing on any discussion of the nature of the contagium in the virus diseases. While engaged in some cross-inoculation studies on certain virus diseases, Johnson (20) noticed that mosaic-like symptoms were secured on tobacco which had been inoculated with juice from apparently healthy potatoes used as controls. This work was repeated, using every care to select potatoes which were perfectly healthy in every way as far as could be determined. The symptoms secured on tobacco plants in this manner were "mottle," "spot-necrosis" and "ring-spot," of which the two former were thought to be different manifestations of the same disease. These "new"

diseases produced in this manner are infectious and can be transmitted from tobacco to tobacco and back to potato, and also have been transmitted to a number of other solanaceous plants. It is difficult to interpret these results. Johnson suggests that they indicate either that potato protoplasm is actually the causal agency of one or more of the tobacco virus diseases or that potatoes are "true carriers" of viruses. It should be stated that these peculiar results are not obtained when seedling potatoes are used for the inoculum.

**Transmission of virus diseases.** — The dissemination of the virus diseases offers a very interesting and useful field of study. Several natural agents of dissemination have been discovered thus far and the disease has been transmitted experimentally in several other ways. The natural agencies known are three: (a) by means of the perennial parts of plants which are propagated vegetatively as in potato tubers, sugar cane cuttings and the underground perennial roots of weeds such as the horse nettle and the milk weed; (b) in seeds as in the bean and the wild cucumber; and (c) by insects as in the potato virus diseases, tomato mosaic, cucumber mosaic, tobacco mosaic, beet curly-top, and many others. Experimentally, virus diseases have been transmitted in several other ways in addition to the three natural agencies. These are: (a) by inoculating the expressed sap from a diseased plant into a healthy plant; (b) by grafting and budding; (c) by the cutting knife and by seed piece contact as in spindle-sprout of potato. There are several ways of making sap inoculations. First, the sap is extracted by pulping and pressing diseased leaves. The virus-carrying juice may be injected by means of a hypodermic needle, or a drop may be placed on a needle prick in a leaf. A very successful means of making inoculations in some of the potato virus diseases is to mutilate the leaf severely and then drop on some virus juice, or simply crush a diseased leaf into a pulp and rub a healthy leaf with some of the pulp held between the thumb and finger until the healthy leaf is well mutilated. In grafting, the simple graft is successful in some cases, while in others inarching is more successful. In certain potato viruses cutting a plug out of a healthy tuber with a cork borer and inserting a plug cut from a diseased tuber has transmitted the disease. A disputed point is that of the possibility of dissemination through the soil. This question will in all probability be answered sooner or later.

**Note.** — For laboratory exercise on virus diseases, see following virus diseases of the peach.



## REVIEW QUESTIONS

1. Distinguish the virus diseases from the ordinary parasitic diseases and from the non-parasitic or so-called "physiological" diseases.
2. What different types of symptoms may be associated with the virus diseases in general?
3. What is meant by masking of symptoms?
4. Name and describe the various theories that have been advanced as to the cause of the virus troubles.
5. Describe the known properties of the virus in those cases where it has been studied extensively.
6. According to Duggar and Karrer, what is the size of the infective particles in the virus? How did they determine this?
7. Discuss the manner of transmission of virus diseases.

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### Virus Diseases of Potatoes

At the outset it is recognized that the large number of different types of virus troubles found on potatoes and the present imperfect state of our knowledge of the whole problem will render any discussion of this

subject which can be given here more or less inadequate and unsatisfactory. At the same time there are many interesting and important facts which are fairly well established, even to such an extent as to give promise of satisfactory control in case of many of the potato virus diseases. In view of these facts, and since the potato is so universally grown and the virus diseases are so generally present, it is felt that at least a brief discussion of some of the more important virus troubles of potatoes is justified.

A type of virus disease of potatoes generally designated as mosaic was observed in Maine in 1912. Apparently the same or similar troubles had been known in Europe under various names for a much longer time. During recent years, since diseases of the virus type have been receiving more intensive study by plant pathologists, there has been an extensive segregation of these troubles into many different types of the malady, each with its own distinct, specific virus capable of initiating the particular set of symptoms with which it is associated. In 1914 there were only three types of disease in potatoes that were recognized as being of the virus type. Orton (16) designated these as leaf-roll, curly-dwarf and mosaic. In 1923 Schultz and Folsom (22) described seven different and distinct types of virus diseases in potatoes, namely, mild mosaic, leaf-rolling mosaic, rugose mosaic, streak, leaf-roll and net-necrosis, spindle-tuber, and unmottled curly-dwarf. By 1925 this list of seven diseases had more than doubled. At the present time upwards of twenty different virus diseases of potatoes have been named by various workers in America and Europe. The list includes, in addition to the seven named above, the following: super-mild mosaic, weather mottling, crinkle mosaic, witches' broom, yellow dwarf, yellowtop, giant hill, calico, Johnson's new virus, intervenal mosaic and marginal leaf-roll. Some of the diseases in this list, of course, are placed there tentatively until further investigation can determine their status. It is not at all unlikely that still other forms of virus diseases will ultimately be found upon the potato. The nomenclature, too, in many cases at least, is tentative and will have to be stabilized either by usage or by general agreement. It will be impossible to discuss all of these different types of potato viruses in the scope of this work but a few of the more important will be selected for further discussion and the others mentioned only in a general way or incidentally. Perhaps the types which are of most general interest from an economic standpoint are rugose mosaic, leaf-roll, spindle-tuber, and mild mosaic, although streak and yellow dwarf are very violent in their reaction when they occur. No one type seems to be equally prevalent in all regions. Leaf-roll is reported to be the most serious of all the potato virus troubles in Indiana, while in Nebraska

spindle-tuber is considered of great economic importance. In western Oregon rugose mosaic is the most prevalent and harmful type of potato virus disease known at the present time.

**Symptoms.** — Since the causal agency of the virus diseases has not yet been identified and classified the only means of distinguishing be-

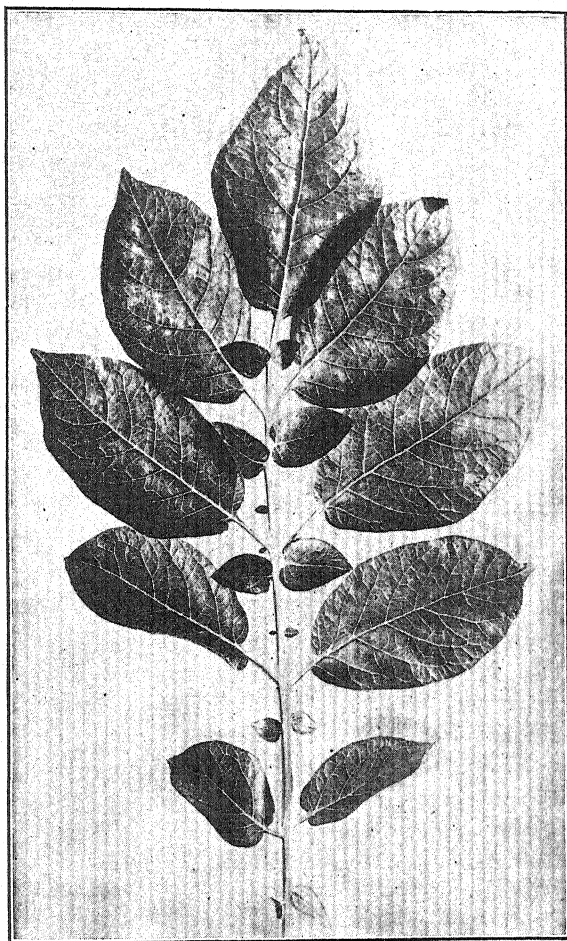


FIG. 199. — Potato leaf showing symptoms of mild mosaic. (Photograph by McKay, Ore. Agr. Exp. Sta.)

tween these different virus diseases is that of the symptoms exhibited. As has been previously mentioned the symptoms are frequently masked by unfavorable environmental conditions, therefore, it becomes necessary to determine these distinguishing characteristics under very care-

fully controlled experimental conditions. Schultz and Folsom (22) have worked out the symptomatology of the potato virus diseases in great detail. The elementary characteristics they consider as "unit symptoms" and combinations of these units are "symptom-complexes." The elementary unit symptoms observed in the various potato diseases of this type are dwarfing, chlorosis, mottling, wrinkling, rugosity, ruffling, curling, rolling, rigidity, necrosis, leaf dropping and premature death. Dwarfing as a symptom is manifest by the general reduction in size of parts, both in length of shoots and petioles and in leaf area. Chlorosis consists of a yellowing of the leaf due to deficiency of chlorophyll. Mottling occurs when the chlorosis is in localized spots. Rugosity consists of an unevenness of the leaf blade surface in which the depressions run along the veins with the mesophyll between the veins uniformly elevated. In wrinkling the surface of the leaf is thrown into elevations and depressions without uniformity. Ruffling is due to an unevenness in which ridges passing from the mid-rib to the leaf margin become more prominent as they approach the edge of the leaf thus producing a ruffling of the margin. Curling is a term applied to the condition resulting when the leaf blade bends downward along the mid-rib. Rolling results from an upward curving of the sides of each leaflet parallel to the mid-rib so as to form a sort of trough. Rigidity and brittleness occur in stems and petioles. Necrosis is manifest by the death and turning brown of certain tissues and may appear in spots or streaks. Leaf dropping occurs in certain cases where whole leaves, beginning with the lower ones, die and drop off. Experience has taught that certain of these unit symptoms are frequently grouped and when a uniform and rather constant grouping is found, this symptom-complex is considered characteristic of a particular type of virus disease. This complex of symptoms is checked up carefully under controlled experiments before it is recognized as the mark of identity of a certain disease. It should be kept in mind that the symptoms of any particular type of virus disease are not exactly the same and do not occur with the same severity on all varieties of potatoes. Neither are the symptoms identical on the same variety under all environmental conditions. When making comparative studies of the different virus diseases of potatoes they should all be studied on the same variety of potatoes and under similar conditions. Following are a number of the potato diseases with the grouping of unit symptoms making up the symptom-complex which is considered sufficient in each case to distinguish that particular type of potato virus disease from all others. The descriptions of symptoms are taken largely from the work of Schultz and

Folsom and apply particularly to characteristics as exhibited by the Green Mountain variety with which they worked.

*Mild mosaic.* — The combination of unit symptoms making up the complex of symptoms which distinguishes this type from all others

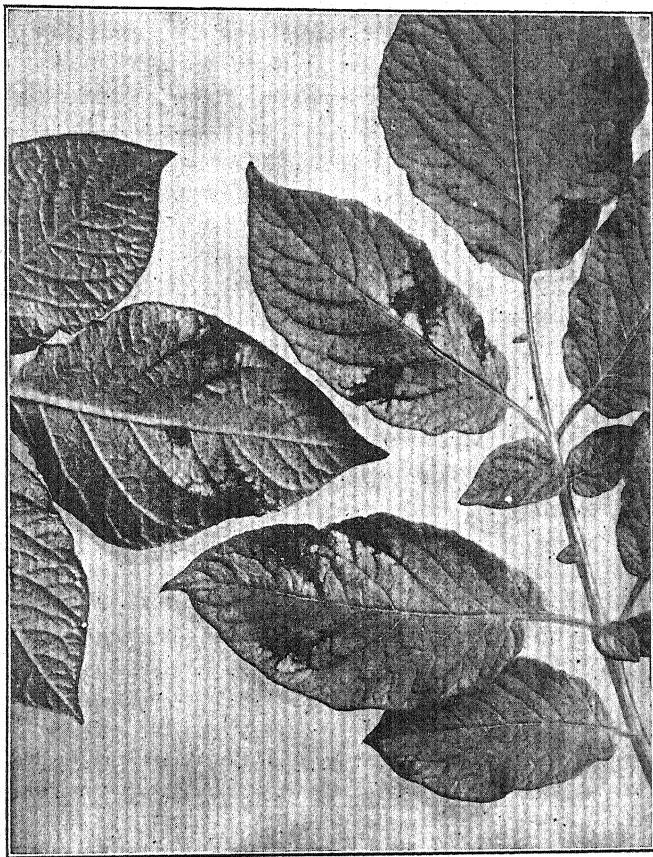


FIG. 200. — Current season symptoms of rugose mosaic. (Photograph by McKay, Ore. Agr. Exp. Sta.)

includes slight dwarfing, distinct mottling, wrinkling, and some ruffling (Fig. 199). There is some reduction in the average size of tubers from diseased plants. Mild mosaic can be transmitted by grafts, aphids and by leaf mutilation.

*Rugose mosaic.* — The unit symptoms making up the total symptom complex in this case are distinct dwarfing, more chlorosis and more

diffuse mottling than in mild mosaic, rugosity, brittleness, both spot and streak necrosis, leaf-dropping and premature death. A striking reduction in the size of the tubers is caused by this disease. The first symptom to appear after inoculation with the virus of rugose mosaic is almost invariably a streak necrosis on the leaves (Fig. 200). In case infection occurs early and the plant makes sufficient growth after infection, all of the other symptoms of typical rugose mosaic (Fig. 201) may appear before the end of the current season. On the other hand, if

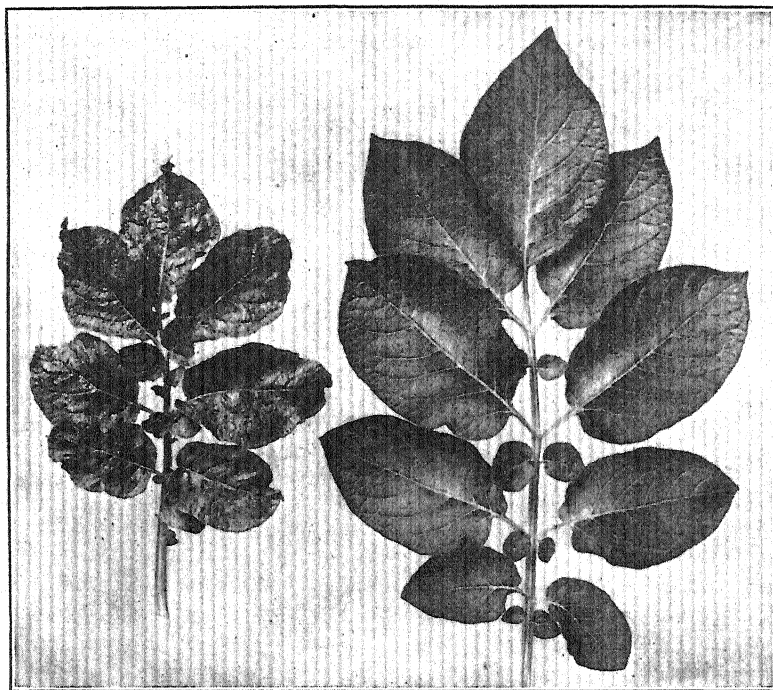


FIG. 201. — Rugose mosaic of potato. Leaf at left, diseased; at right, healthy. (Photograph by McKay, Ore. Agr. Exp. Sta.)

infection occurs toward the end of the growing season, only some of the earlier symptoms, as streak necrosis or leaf-fall, may appear during the current season, the later-appearing symptoms not showing up during the current season but appearing on the progeny the next year. Rugose mosaic has been transmitted by aphids and by leaf mutilation.

*Leaf-roll.* — The characteristic symptoms of leaf-roll are uprightness, rigidity, rolling of the leaves, dwarfing, chlorosis and necrosis. The striking characteristic of the disease is the stiff uprightness of the plant with the lateral margins of the leaflets rolled upward (Fig. 202). In a



microscopic examination of sections of the stem, phloem necrosis is a valuable diagnostic character. Evidence of necrosis may sometimes appear in the tubers also. Tuber yield is reduced but the extent to which this occurs is difficult to estimate. Leaf-roll is difficult to transmit. This has been accomplished only by grafts and by aphids, leaf mutilations proving unsuccessful.

*Spindle-tuber.* — The tuber symptoms associated with this disease are abnormal spindliness, cylindrical shape and conspicuous eyes. Symptoms are manifest on the aerial parts also. The plants tend to be



FIG. 202. — Potato leaf-roll. (Photograph by McKay, Ore. Agr. Exp. Sta.)

spindling, upright, often darker green in color and with slightly rugose leaves. There is an absence of mottling, leaf-rolling and streak. The tuber yield is reduced. Spindle-tuber can be transmitted by grafts, aphids, leaf mutilation, the cutting knife, and by seed piece contact.

*Streak.* — While this disease is not now known to be widely distributed it is included here as an example of a very violent virus disease. The current-season symptoms include streaking and spotting, burning, brittleness, leaf dropping, and premature death. Generally no mottling or wrinkling occurs. The symptoms on the tubers are extreme reduction in size, cracking or splitting, and darkening near the eyes. Tuber formation is so suppressed that the disease is usually not tuber perpetu-



ated for more than a year or two and soon runs out. If the disease is tuber perpetuated in the few small weak tubers that mature, the next year's symptoms are extreme resulting in premature death and the running out of the stock.

**Control.** — Several facts are known about the virus diseases of potatoes which must be taken into consideration in devising control measures. (a) In all cases the virus is transmitted from generation to generation in the tubers, that is, unless and until tuber formation is

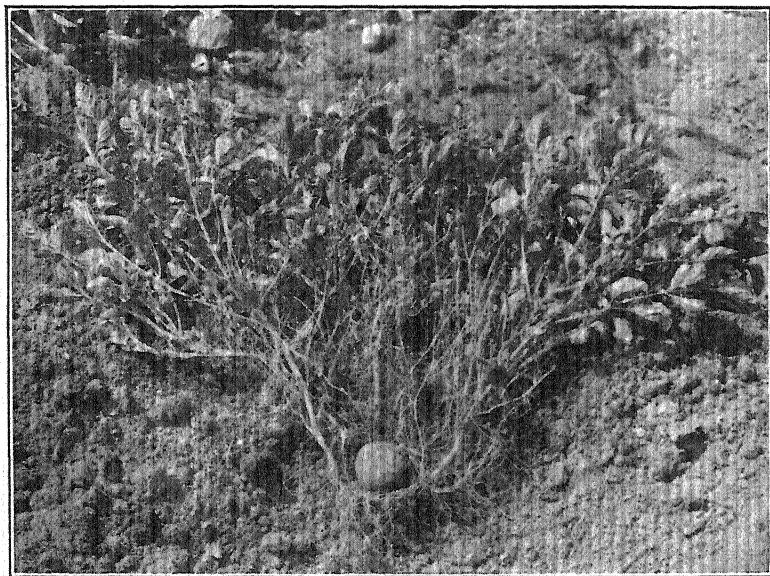


FIG. 203. — Witches' broom of potato. See reference 12. (Photograph by McKay, Ore. Agr. Exp. Sta.)

entirely inhibited. (b) In practically all cases that have been carefully investigated aphids are known to be carriers of the virus. (c) In at least one type of disease the virus is transmitted on the seed-cutting knife and by seed piece contact. There has been a difference of opinion as to whether the virus is transmitted through the soil. In Europe the opinion that it can be transmitted in this manner has persisted for many years. In the United States the opinion has quite generally prevailed that there is no definite proof of soil transmission, although recently this conviction has been weakened somewhat. It can readily be seen, then, that in the light of present knowledge the two chief points of attack will be centered on insect transmission and tuber perpetuation, with some attention given to the possibility of transmission by the cutting

knife and by seed piece contact in the case where this method of transmission has been demonstrated.

**Insect control.** — Theoretically the control of insects would largely prevent the transfer of the viruses from diseased to healthy plants in the field, unless it should be proved that they persist in the soil. There is undoubtedly a considerable spread of potato virus diseases in the field due to aphids. If the aphids could be controlled there would probably be very little transmission of potato viruses in the field. In actual practice, however, aphids are very difficult to control. Thus the problem of controlling potato virus diseases by insect control becomes a difficult one. Since potatoes are usually sprayed with bordeaux for fungous diseases, it has been recommended that black-leaf-40 (nicotine sulfate) be added to the bordeaux spray at the rate of three-fourths pint of the nicotine to each fifty gallons of the spray. This treatment may keep the aphids somewhat in check but it is doubtful if it will be entirely effective in preventing the spread of potato virus diseases in the field.

**Eliminating diseased tubers and plants.** — Attacking the virus-disease control problem through insect control is at best an expedient which should be subordinate to the problem of securing virus-free seed stock. The real fight on virus diseases of potatoes, it would seem at present, will necessarily be concerned with eliminating the diseases from the seed stock. At present the two chief methods available for thus ridding a strain of potatoes of virus diseases are (a) field roguing, and (b) tuber or eye indexing. Successful field roguing depends upon the ability to detect diseased plants through the symptoms exhibited. Since, as has been previously indicated, the symptoms are frequently more or less masked because of environmental conditions, it is very difficult and many times impossible to eliminate all disease by field roguing. Nevertheless this method should be vigorously followed up and every diseased plant detected should be ruthlessly rogued out.

**Tuber indexing.** — During the last few years, as the result of attempts to overcome the uncertainties of field roguing, a system of tuber indexing has been developed which gives great promise in building up stocks of disease-free seed tubers. This system consists essentially in selecting one tuber from a hill and planting it early, either in the greenhouse or out of doors. When this plant has developed far enough for symptoms to be evident it is examined for indications of virus diseases. If it proves to be healthy all the other tubers of the same hill are assumed to be free from viruses and are used for seed purposes. If the index tuber produces a diseased plant all the other tubers from the same hill are discarded. The success of this method depends upon growing the

index tubers under favorable conditions for bringing out clearly all the symptoms. In the greenhouse proper temperatures and other growing conditions can be maintained so that full expression of symptoms can be obtained. For practical field work the planting of the index tubers in April will generally afford the proper weather conditions for showing up most of the virus diseases so that the index plots can be read in time to plant the main crop in June. This system is especially recommended in selecting seed for the special seed plot where seed is to be grown for the commercial planting the next year. One flaw occasionally develops in this system. Sometimes when infection occurs late in the season just before the plants mature the virus does not become thoroughly distributed to all parts of the plant before growth ceases. In such cases it may happen that some of the tubers in a single hill will carry the virus while others will be free from disease. In tuber indexing such a hill, if by chance one of the virus-free tubers should be chosen for germinating, the index would indicate a healthy hill when in reality a certain percentage of the tubers in that hill would be virus-carriers. In a case of this kind the only way to know definitely concerning the disease status of all tubers in the hill would be to eye index each tuber. This is done by cutting one eye out of each tuber and sprouting it until the symptoms can be read. This, of course, is a very tedious and expensive operation but can be used effectively on a limited scale when necessary to make absolutely certain that every tuber planted is free from virus disease. In most cases, however, this extreme eye-indexing procedure is not necessary and the indexing of one tuber from each hill would insure a reasonable freedom from virus disease. This indexing method should be followed up by roguing in the field to eliminate any scattered diseased plants that perchance might not have been discarded in the indexing process. The roguing should be started as soon as the first leaves appear above ground.

*Special seed plot.* — As is true with some of the fungous diseases of potatoes, the special seed plot is also a valuable adjunct in controlling the virus diseases. Because of the danger of insect transmission the seed plot should be isolated at some distance from any other potato fields (Fig. 9). If the seed planted in this special plot is carefully indexed before planting each year and the plot is rogued thoroughly at intervals throughout the season there should be no difficulty in growing a supply of clean seed each year, especially if the plot is completely isolated from other potato fields and the aphids are controlled by proper spraying to prevent any current season infections. An important and necessary aid to successful roguing is the practice of tuber unit planting. This method consists in planting all seed pieces from each tuber in

consecutive order in the row and marking the limits of each tuber. In roguing, then, if any single sprout shows mosaic symptoms, all the hills grown from that tuber unit should immediately be taken out whether or not all of them exhibit the mosaic symptoms. This insures a much cleaner seed plot than could otherwise be maintained because where the tuber unit system of planting is not practiced the most careful roguing can not attain nearly the efficiency in eliminating diseased plants that is reached where the tuber unit system is used.

#### REVIEW QUESTIONS

1. Name at least fifteen different virus diseases which have been described on the potato.
2. How do Schultz and Folsom use the terms "unit symptoms" and "symptom complexes"? (See text and reference 22.)
3. What are the elementary unit symptoms observed by the above workers in the several virus diseases of potatoes studied by them?
4. Describe the symptoms of five of the more important virus diseases occurring on potatoes.
5. How are potato mosaics transmitted from generation to generation?
6. What agencies may be responsible for field dissemination of potato virus diseases?
7. Mention the various possible means of combating virus diseases of potatoes.
8. What difficulties are met in attempting to control these diseases by roguing?
9. Describe the process of tuber indexing and eye indexing.
10. What place has the special seed plot in the campaign against potato viruses?

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### Cucurbit Mosaic

This disease of cucumber and other cucurbits has now been known with certainty for some ten or fifteen years, but a malady which probably was mosaic was reported from certain localities at the beginning of the present century. In 1902 Selby (11) reported a disease on greenhouse cucumbers in Ohio which he thought resembled the mosaic disease of tobacco and tomato. He mentioned a yellowing of the leaves and a

tendency to unfruitfulness. In 1910 Stone (12) of Massachusetts called attention to a disease of cucumbers and muskmelons, which had been observed in that state for a number of years. He stated that it caused a mottling of the leaves similar to tobacco calico or mosaic. Clinton (1) reported a disease on muskmelon in Connecticut in 1909 and suggested that it resembled the trouble described by Selby in Ohio several years previously. He suspected that it might have spread from tobacco to the melons since the land had grown tobacco the previous season. In a later report Clinton (2) noted the occurrence of mosaic or white pickle disease which was observed on cucumbers in Connecticut in 1914. In this case the crop was practically ruined at the time it should have come into full bearing. Coons (3) reported that the disease was first called to the attention of the Michigan Experiment Station workers in 1912 on specimens from Grand Rapids in that state. He mentions a mottling of the leaves, shortening of the main shoots, and the warty, roughened, appearance of the fruits. Evidence of growers indicates that the trouble had been present for several years. Indications pointed to the infectiousness of the disease but this was not demonstrated. The really serious investigation of this malady was taken up about 1915 when Jagger and Doolittle began work simultaneously, the former in New York, working with greenhouse cucumbers, and the latter working in the field in Michigan. Jagger (9) carried out an extensive set of inoculation experiments, establishing beyond doubt the infectious nature of the disease, both by the use of infected juice and by insect transmission. Doolittle (4) carried out a similar set of inoculation experiments in the field. He secured infection by injecting sap; tearing off leaves of a healthy plant and touching the wound with the broken end of a petiole from a diseased plant; inserting pieces of diseased vines into slits made in healthy stems; and by means of aphids. In 1918 Jagger (10) reported the results of cross inoculation experiments in which he showed the susceptibility of species and varieties in eight different genera of the Cucurbitaceae and two genera in other families. In 1920 Doolittle (6) published in bulletin form a complete account of this disease including all the known facts up to that time. Recently Doolittle and Walker (7, 13) have published additional facts concerning the overwintering of the cucurbit mosaic and certain new hosts for the virus.

At present the disease is widely distributed in the United States. In 1916 the disease had been reported as present in greenhouses in Minnesota, Michigan, New York, Connecticut, Pennsylvania, Illinois, Indiana, Ohio, and Louisiana. Reports of its occurrence in the field up to the same date came from Wisconsin, Michigan, Indiana, Ohio,

Iowa, Illinois, Vermont, New York, Minnesota, Massachusetts, Virginia, and Ontario, Canada. In 1920 the number of states in which the disease was known either in the greenhouse or field had been increased by the addition of the following: Colorado, Florida, Maine, New Jersey, West Virginia, Georgia, Texas, Nebraska and California.

**Susceptible species and varieties.** — In view of recent work on differential hosts for mosaic diseases any discussion of cross inoculation work with the viruses must take into consideration the fact that results must be more or less tentative until the specificity of all the different viruses has been determined and the host ranges for each definitely established. The cucurbit virus, however, seems to be fairly well stabilized so that the following host relationships can be accepted with a considerable degree of assurance. As a result of the work of Jagger (10), Doolittle (6, 7) and Walker (13) the following extensive list of species has been proved susceptible to the cucurbit virus. In the family Cucurbitaceae there are 12 genera and not less than 24 species, namely, *Cucumis sativa* (over 25 varieties of cucumbers), *Cucumis melo* (several varieties of muskmelons), *Cucumis anguria* (bur cucumber or West Indian gherkin), *Cucumis grossulariaeformis*, *Cucumis metuliferus*, *Cucumis odoratissimus*, *Cucumis ficifolia*, *Cucurbita moschata* (pumpkin), *Cucurbita pepo* (pumpkin), *Cucurbita pepo* var. *condensa* (squash), *Cucurbita maxima* (gourd), *Lagenaria vulgaris* (gourd), *Lagenaria leucantha*, *Luffa cylindrica* (dishcloth gourd), *Luffa acutangulis*, *Benincasa cerifera*, *Benincasa hispida*, *Bryonopsis laciniosa*, *Ecballium elaterium*, *Micrampelis lobata*, *Momordica charantia*, *Momordica involucrata*, *Melothria scabra*, *Sicyos angulatus*, and *Trichosanthes anguina*. In addition to these species of the Cucurbitaceae a number of species belonging to other families have been found susceptible. These include: *Helianthus debilis* (cucumber-leaved sunflower), *Lobelia erinus*, *Asclepias syriaca* (milkweed), *Phytolacca decandra* (pokeweed), *Nepeta cataria* (catnip), *Myrtinia louisiana*, *Amaranthus retroflexus* (pigweed), *Cap-sicum annuum* (pepper), *Physalis pubescens* (ground cherry), *Physalis subglabrata* and *Physalis heterophylla*.

**Economic importance.** — Since the first discovery of cucurbit mosaic, it apparently has rapidly increased in prevalence and importance. Although the disease occurs on muskmelons, squashes, and pumpkins as well as on cucumbers, the greater part of the loss occasioned is sustained by the cucumber growers. Considerable damage is also inflicted upon muskmelons in some sections of the country. The disease has naturally become more widespread and severe in those regions where large acreages of cucumbers are grown under intensive cultivation for the pickle industry. In many such localities mosaic is considered one

of the serious diseases of this crop and extensive losses are sustained. In 1923 The Plant Disease Reporter (Supplement 34 : 204) estimated a loss of 15 per cent in Wisconsin. One field in Illinois showed 50 to 70 per cent mosaic, and extensive losses ranging from 25 to 60 per cent were reported in greenhouses in the Chicago district. Losses of cucumbers from mosaic in 1924 (Supplement 41 : 267) were estimated as follows: Wisconsin 20 per cent; New York 10-20 per cent; Iowa 15 per cent; Delaware 5 per cent; Illinois 3 per cent; and Pennsylvania 2 per cent. On Long Island, N. Y., mosaic reduced the pickle crop 75 per cent. Some growers abandoned pickle growing because of mosaic. In 1925 The Reporter (Supplement 45) reported the cucumber mosaic from seventeen states east of the Great Plains. Losses were reported as follows: Kentucky 20 per cent; West Virginia 10 per cent; Iowa 5 per cent; and New York 3 per cent. No figures are given on which to base a monetary valuation for these losses, but it undoubtedly is considerable.

**Symptoms.** — All parts of the plant may show more or less conspicuous symptoms, but the effects of the disease are most pronounced on

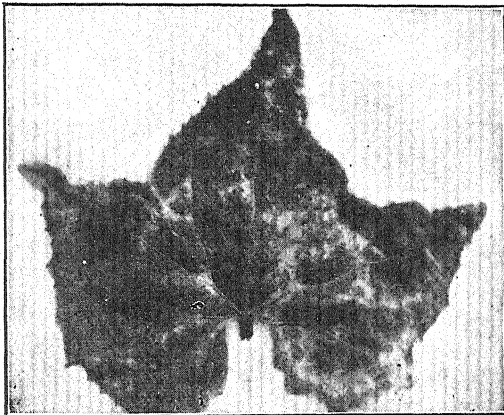


FIG. 204. — Cucurbit mosaic on cucumber leaf. (After Doolittle, U. S. Dept. Agr. Bul. 879.)

the foliage and fruits. The stems show conspicuous modifications also due to the disease. The plants are usually affected when a few weeks old and in a vigorous growing condition, although they may become infected in the very young seedling stage or after the vines have reached old age. Doolittle (6) has given the most complete discussion of the symptoms. The following descriptions apply particularly to the



cucumber, although the symptoms on the other cucurbits are quite similar in many respects.

*On the leaves.* — If infection occurs in the young seedling stage, the cotyledons turn yellow and wilt and the young leaves are slightly mottled. On vigorously growing plants several weeks old, the youngest leaves develop small greenish-yellow areas, circular or angular in outline and limited by small veins. These spots are slightly more translucent than the remainder of the leaf. Sometimes these spots can not be seen well except by transmitted light. Occasionally the sharply defined yellow areas are lacking and the tip of the leaf changes from normal green to yellow accompanied by a gradual downward curling of the edges of the leaf. The surface of the leaf becomes finely wrinkled. After these preliminary symptoms, more pronounced effects appear and the leaf becomes strikingly mottled (Fig. 204) and all subsequent growth is much dwarfed. New leaves may be dwarfed to one-half natural size, and are much more distinctly wrinkled and have much shortened petioles. The green and yellow areas vary in size in different cases. Sometimes the areas are comparatively small, while in other cases there are a few larger yellow patches scattered over the leaf. In the older leaves, the yellowing frequently tends to spread over the entire leaf. On mature plants the older leaves gradually die and drop off.

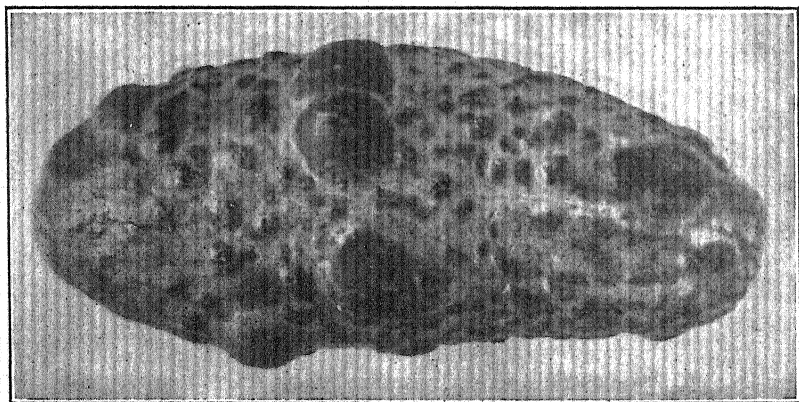


FIG. 205. — Cucurbit mosaic on cucumber fruit. (After Doolittle, U. S. Dept. Agr. Bul. 879.)

*On the fruits.* — A yellowish green mottling first appears on the stem end of the fruit. Ordinarily the mottling gradually spreads over the entire fruit until it becomes a light yellowish-green with darker green spots scattered over the surface. The dark spots are usually elevated above the yellowish area giving the pickle a warty, distorted appearance

(Fig. 205). The mottling may occur in the absence of wart formation, especially in the greenhouse. Sometimes, in later stages of the disease, fruits are produced which are smooth and pale whitish green in color, giving rise to the term "White pickle" which has been applied to the disease by growers.

*Stem symptoms.* — Probably the most evident symptoms on the stems is the decided stunting or shortening effect. The stem internodes are shortened giving a bunched or bushy appearance. On older plants, where the old leaves have died, the stem becomes whitish and seems more brittle. There is no mottling on the stem but it often becomes a yellowish-green in color.

*On other parts.* — The blossom symptoms are not striking and consist chiefly in dwarfing and a slightly paler coloration than normal. No external symptoms show on the roots except a reduction in the number of finer rootlets in the later stages of the disease.

*Transmission.* — In nature there are two general agencies of perpetuation and dissemination. (a) The virus hibernates in certain perennial plants and in certain seeds. (b) Certain species of insects are instrumental in disseminating the virus during the growing season. It has now been proved that the cucurbit virus will infect several species of perennial weeds where it lives over in the underground dormant parts and becomes evident again the next spring when these weeds send up new growth. The perennial weeds thus far determined as hosts for this virus are: *Asclepias syriaca* (milkweed), *Phytolacca decandra* (pokeweed), *Nepeta cataria* (catnip), *Physalis subglabrata*, and *P. heterophylla* (perennial ground cherry). Only one case of seed transmission has been discovered. The seeds of the wild cucumber (*Micrampelis lobata*) have been proved to carry the cucurbit virus. All efforts to demonstrate that the virus is carried in seeds of the cultivated cucurbits have failed.

After the virus has overwintered in these weeds it is an easy matter for insects to carry it to the cultivated cucurbits. Three species of insects have been implicated in this dissemination, namely the cucumber aphid, and the striped and the 12-spotted cucumber beetles. According to Doolittle, the striped beetle is the chief agency in carrying the virus from the wild cucumber to the cultivated forms in the spring. Experiments indicate that these insects may thus carry the disease over distances of 400 yards at least.

Aside from these natural agencies, the cultural operations necessary in growing cucurbits probably play an important part in spreading the disease once it gets a start in a field. Any form of handling which breaks or bruises the plant offers an opportunity for transmitting the disease

from plant to plant. In harvesting the fruit, juice undoubtedly is transferred from plant to plant. In training the vines where they overlap or entwine together, more or less breaking and rubbing together of parts occurs. Workmen walking through the vines trample them. There is strong evidence that the disease is disseminated in all these ways.

**Control.** — In view of the above facts there are four chief lines of attack in combating this disease, namely: (a) destruction of the weed hosts; (b) control of the insects responsible for its dissemination; (c) roguing out diseased cucurbits; and (d) sanitation. Since the virus is known to be perpetuated in several species of perennial weeds this phase of the control problem becomes a difficult one. The milkweed, ground cherries and pokeweed are very common and widely distributed weeds throughout many of the regions where the cucurbit mosaic prevails. Their perennial nature makes them difficult to eradicate and, since insects carry the virus for at least several hundred yards, these weeds must not only be eradicated from the immediate field in which the cucurbits are growing, but from adjoining fields as well. The weeds often grow abundantly in uncultivated fields such as pastures and woodlots, and this adds to the difficulty of eradication.

The problem of controlling plant diseases through the control of insect disseminators is always a baffling one and has never been solved with complete success. In case of cucurbits grown in the greenhouse both insect carriers and diseased plants can be successfully eliminated, but in the field the problem is far more difficult. A few plants can be caged to keep away the insects, but this is hardly practicable on a large scale. A spray of bordeaux mixture plus lead arsenate has been used against cucumber beetles with some success (See under Cucurbit Wilt, p. 211).

*Roguing and sanitation.* — All diseased plants should be rogued as soon as they are detected. Diseased cucurbits are as much a menace as the weed hosts referred to above. In roguing out diseased cucumber plants not only is the source of inoculum for insects removed, but the danger of spreading the disease through the various cultural operations is also lessened. This latter agency of disseminating the disease, of course, is much more apt to spread it among the cultivated cucurbits than from the weed hosts to the crop plants.

#### REVIEW QUESTIONS

1. Describe the symptoms of cucumber mosaic.
2. Name the plants, both cultivated and wild, which are known to be susceptible to this disease.
3. Where does the virus hibernate?
4. How is dissemination effected?
5. Discuss control measures.

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Curly-top of Beet

This disease has been known under various names in the western United States for over a quarter of a century. One of the first names applied to the trouble when it was recognized as a distinct disease was "California beet disease." It has since been referred to as western blight, stunted beets, whiskered beets, hairy-root, curly-leaf, and curly-top. The last mentioned name is now in common use to designate this malady. At first the curly-top disease was probably confused with other types of diseases or injury but for a long time it has been recognized as a sugar beet disease entirely distinct from all other troubles on that crop. There is strong evidence, however, that confusion still exists, or at least has existed until quite recently, over certain troubles on other crops which now seem to be due to the curly-top virus.

**History and distribution.** — The beet curly-top has been recognized as a distinct disease in the western United States since about 1900. Severe losses due to this trouble occurred in California in 1899. In 1902 Townsend (17) described and figured the malady. In 1906

Ball (1) stated his belief that the beet leaf-hopper bore some causal relation to the disease. The next year he reported further evidence that attacks of the leaf-hopper are related to curly-top. In 1907 Smith (13) expressed the opinion that beet curly-top might be similar in nature to the tobacco mosaic. In 1908 Townsend (18) published an extensive account of curly-top, describing the symptoms and occurrence of the disease and suggesting several possible causes of the trouble but arriving at no definite conclusion on that point. Ball (2, 3) published

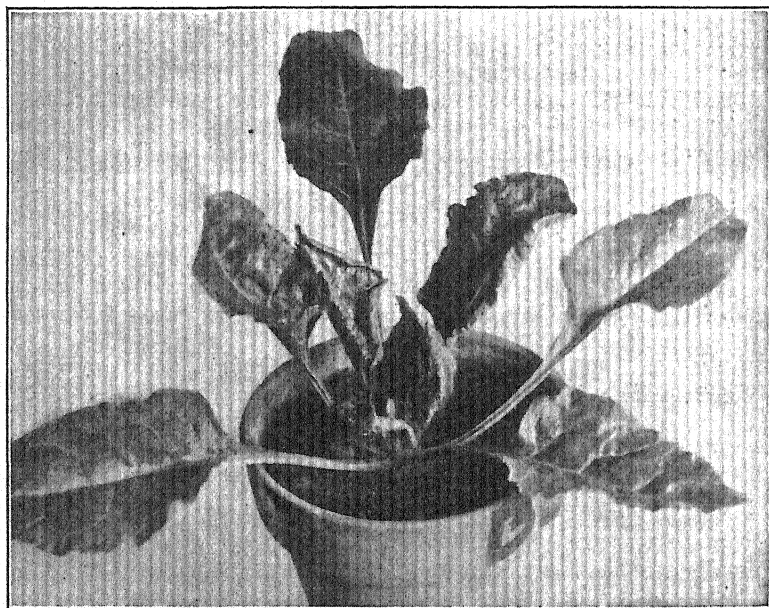


FIG. 206. — Curly-top of sugar beet. (Photograph by McKay, Ore. Agr. Exp. Sta.)

further evidence in 1909 and in 1917 to substantiate his claim that the beet leaf-hopper is responsible for the transmission of the disease. In 1915 Smith and Bonquet (14) reported on the length of time necessary for hoppers to feed on beets in order to transmit the disease. They found that five minutes was a sufficient length of time for transmission to occur in this manner but that it might require about two weeks for the symptoms to appear on the infected plant. In a later paper (15) the same writers confirm the statement of Bonquet and Hartung (4) that leaf-hoppers must previously feed upon curly-top beets before they can transmit the disease to healthy plants. In 1917 Bonquet and Stahl (5) reported the occurrence of curly-top in certain native plants.

Further substantiation of the claim that leaf-hoppers must feed upon diseased plants before being able to transmit the virus was made by Stahl and Carsner (16) in 1918 when they demonstrated that recently emerged hoppers, if lifted from the plant before feeding, did not transmit the disease. In 1919 Carsner (6) published a list of 14 species of susceptible plants other than sugar beets. Still later, in 1924, Carsner and Stahl (7) published the results of extensive studies of various phases of the trouble, including distribution, hosts, economic importance, environmental factors, incubation period and overwintering of the virus.

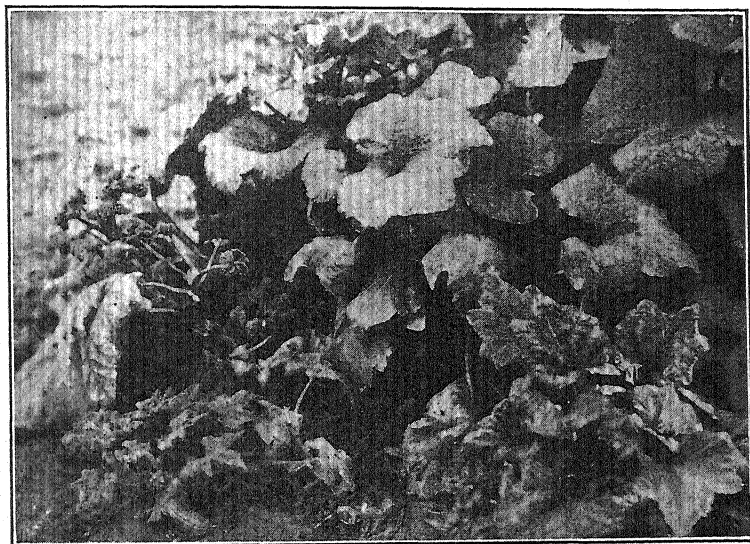


FIG. 207. — Curly-top symptoms on squash. (Photograph by Arthur Bowman of H. A. Hyde Co., Portland, Oregon.)

At the present time the curly-top disease occurs in the arid or semi-arid regions of western North America. Its distribution is limited to the regions where the leaf-hopper, *Eutettix tenella* Baker, is found. It has been reported from the states of Arizona, California, Colorado, Idaho, Kansas, Nebraska, New Mexico, Oregon, Texas, Utah, and Washington. There are also recent reports of the limited occurrence of this disease on beets or other plants in isolated cases as far east as the Mississippi River.

**Hosts.** — During the course of investigations since curly-top has been known, the range of hosts has been gradually extended. At first, attention was turned toward the discovery of possible wild hosts because of the bearing these might have on overwintering. Among the

wild hosts now known are: red-stem filaree (*Erodium cicutarium*), chickweed (*Stellaria media*), tumble-weed (*Amaranthus graecizans*), pigweed (*Chenopodium album*), bur clover (*Medicago hispida*), wire grass (*Polygonum aviculare*), and small nettle (*Urtica urens*). Among the cultivated hosts are buckwheat and spinach, reported in 1916 (6).



FIG. 208. — Curly-top symptoms on beans. (Photograph by Arthur Bowman of H. A. Hyde Co.)

Tomato was listed as susceptible in 1924 by Carsner and Stahl (7), and in 1926 Carsner (8) reported the bean as susceptible to the curly-top virus. During the season of 1926 McKay and Dykstra (9, 10) made observations in Oregon which indicated that squash and tomato are subject to attack by the curly-top disease. They demonstrated by



leaf-hopper inoculations that the western yellow tomato blight, the cause of which has baffled plant pathologists for many years, is due to the curly-top virus. In 1927 Severin (19) added to this list of hosts the following: alfalfa, pumpkin, watermelon, cucumber, muskmelon, cantaloupe, potato, pepper, cabbage, turnip, radish and horse-radish.

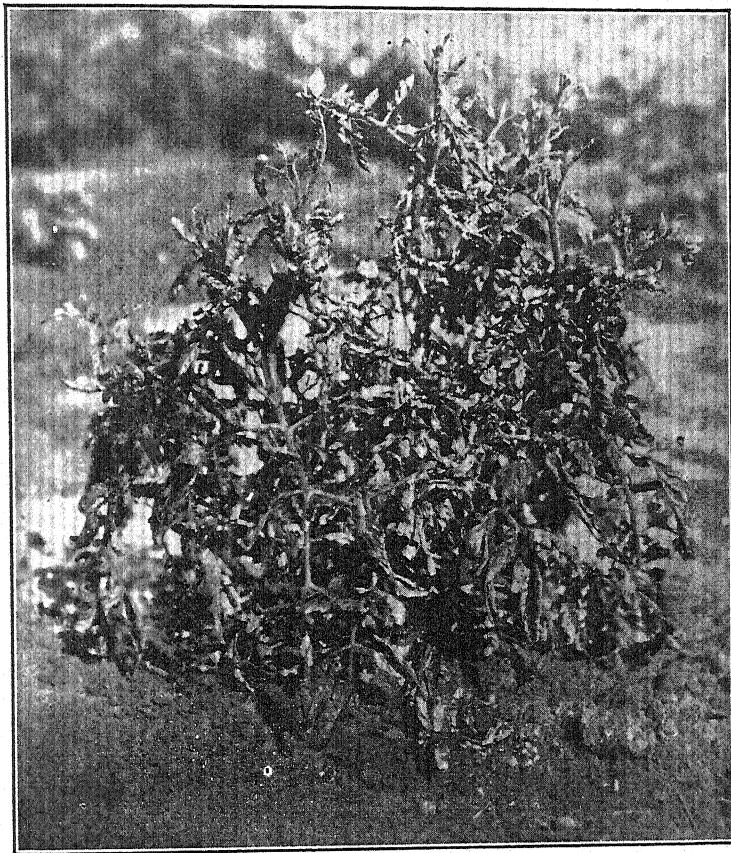


FIG. 209. — Western yellow blight of tomato, now known to be caused by the curly-top virus. (After McKay, Ore. Agr. Exp. Sta.)

**Symptoms.** — The most striking symptoms of curly-top are exhibited by the leaves that develop after the plants become infected. Such leaves are much dwarfed, crinkled and puckered (Fig. 206). An irregular swelling of the veins occurs on the under side of the leaf. The beet roots also show marked symptoms. They are tough and woody, and usually develop hairy or woolly roots. They also show dark rings of fibro-vascular bundles. The symptoms on plants other than beets



are quite similar. On squash, the leaves which are present when infection occurs become yellow and sickly. All new growth of vine and leaves, developing after infection of the plant has occurred, is much dwarfed (Fig. 207).

**Cause.** — Curly-top is caused by a virus which seems to have a biological relationship with the beet leaf-hopper since it apparently is not transmitted in nature except by this insect. The virus has not been transmitted artificially by juice inoculations as has been done with many of the viruses. It apparently is necessary that the virus incubate for 4 to 6 hours in the leaf-hopper before the insect can transmit it to healthy plants. More often this period is 24 to 48 hours. After a viruliferous hopper has fed on a healthy plant it usually requires a week or two before symptoms appear. The shortest period recorded is 4 days. The virus may overwinter in wild hosts such as filaree or chickweed, in volunteer beets or in the insect. Conditions which favor the increase and spread of the leaf-hopper are conducive to outbreaks of curly-top. Under favorable seasonal conditions leaf-hoppers may invade localities where they are not commonly known and cause outbreaks of the disease.

**Control.** — The chief means of prevention that suggests itself is insect control by means of insecticides, repellants or parasites. None of these has as yet proved successful. In the more southerly regions sugar beets may be planted early enough so that they escape the disease until quite large because the hoppers do not appear in the cultivated fields until spring. In southern California beets planted in November or December produced good crops while plantings made in March succumbed to curly-top. There is a possibility that resistant strains of beets may be developed.

#### REVIEW QUESTIONS

1. Describe the symptoms of curly-top on beets.
2. Name some other cultivated plants which have more recently been demonstrated to be susceptible to curly-top.
3. Wherein does the method of transmission of curly-top differ from that of most other known virus diseases of plants? Compare with certain diseases known in the animal kingdom.
4. Name some wild plants thought to be instrumental in the overwintering of curly-top.
5. What is the geographic range of this disease? What determines this range?

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### Virus Diseases of the Peach

There are three definite and distinct diseases of the peach that are beyond doubt of the nature of virus diseases. These are peach yellows, little peach, and peach rosette. Yellows is the oldest, best known and most widely distributed of the three diseases. Yellows has been known for over a hundred years while the other two diseases have been recognized scarcely half that long. Like all virus diseases, those of the peach were of very obscure origin so that when first observed by growers and others, many erroneous theories were advanced as to the cause of the

trouble. The communicability of peach yellows was established before the end of the nineteenth century and both little peach and peach rosette are now known to be transmissible.

### Peach Yellows

Fairly authentic records of peach yellows date back to 1791 and accounts of unhealthy conditions in peach trees which may have been peach yellows would place the date back as early as 1750 to 1760. The peach was extensively grown by the earliest settlers in the United States and was highly prized by them. For many years the trees seemed to flourish and to be remarkably free from diseases. The region along the Delaware River and especially in the vicinity of Philadelphia became famous for its peach orchards and it was in this section of the country that the disease which became known as yellows first attracted attention. Erwin F. Smith (11) has given us an extensive review of the history of peach growing in this country and of the first appearance of peach yellows in those sections of New Jersey, Delaware, and Pennsylvania surrounding the city of Philadelphia. He cites the writings of Judge Richard Peters (10) and others on peach growing as evidence that the disease which is now known as yellows probably made its first appearance in that vicinity soon after the middle of the eighteenth century.

The disease gradually spread from the point of its first appearance. It soon reached New York and Maryland, and by 1815 it had reached Connecticut. The disease apparently spread northward and westward very gradually. It was reported from Ohio in 1849. It is thought to have first appeared in Michigan about 1867. There is some evidence that the disease appeared in Indiana as early as 1842. The disease probably did not occur in Canada previous to about 1870, but by 1880 it was prevalent in certain sections of Ontario. In Virginia the yellows apparently had become established by 1849. The present distribution of peach yellows in the United States extends from the New England States southward to South Carolina and Georgia, and westward to Michigan, Missouri, Kansas and Texas.

This disease affects the peach particularly, but is also said to attack plums, apricots, almonds, and nectarines. In certain peach growing sections it is one of the most dreaded of peach diseases. According to the records the disease appears in cycles, outbreaks occurring periodically. Epidemics occurred in 1791, 1806, 1817 to 1821, 1845 to 1858, 1874 to 1878, and 1886 to 1888. Exact figures on the losses occasioned by this disease are difficult to obtain but there is no doubt that they

have been enormous in the aggregate since the malady first appeared. There are records of the destruction of whole orchards, and peach growing has been abandoned in many communities following serious outbreaks of the disease. In 1888 Smith (11) submitted figures showing the losses in many individual orchards. A few examples will serve to indicate the seriousness of the trouble. In one typical case in Maryland there were 953 dead and diseased trees in a total of 2974 trees in the orchard, or a 32 per cent loss. In a Delaware orchard of 3520 trees, 2616, or 74 per cent, were found affected with yellows. Another orchard in Delaware suffered a loss of 88 per cent of a total of 2146 trees. The only recent figures available are those of The Plant Disease Survey. The Plant Disease Reporter (Sup. 36) estimates the losses from yellows and little peach in 1923 at 75,000 bu. for the four states of New York, New Jersey, Pennsylvania, and Michigan. In 1924 The Reporter (Sup. 43) gave the losses for New Jersey, Pennsylvania, Maryland and Virginia as totaling 98,000 bu.

**Symptoms.** — Certain symptoms usually appear during the first year of attack. These are supplemented by other symptoms which appear in advanced stages of the disease during the second year and later. The complete list of symptoms shown in the advanced stage of yellows includes: (a) premature ripening of the fruit, (b) an abnormal coloration of the skin and flesh of the fruit, (c) the development of dwarfed, wiry, sickly shoots, and (d) a yellowish-green appearance of the foliage. It will thus be seen that both the fruit and the twigs and foliage may exhibit characteristic symptoms of yellows.

*The fruit.* — In typical yellows the fruit matures from a few days to two or three weeks in advance of the usual time of ripening. Smith (11) states that sometimes this period precedes the normal by six weeks or more. All or only a portion of the fruit on an affected tree may act in this way. Usually certain peculiarities of coloration accompany this premature ripening. The normal blush on the skin of the fruit is replaced by red spots or blotches. The spotting or blotching may also extend throughout the flesh of the fruit, and the flesh around the pit is redder in color than normal. Sometimes diseased fruits have an insipid or bitter flavor, or again the taste may be normal. These fruit symptoms appear the first season, and also the next year with increased severity. Usually the tree is worthless for fruit production after the second year and sometimes after the first year of attack.

*The twigs and foliage.* — The most striking twig symptom consists in the development of many slender, wiry, finely branched twigs from dormant buds on the trunk and branches. These growths are frequently not unlike the witches' brooms caused by various fungi. This

abnormal twig growth may appear during the first season of attack, or the prematuring of fruit may occur for one or more seasons before the twig symptoms appear. The leaves on these abnormal shoots are usually very narrow and the whole shoot looks sickly. A general yellowish-green appearance of the foliage is a frequent accompaniment of yellows, but since this symptom may be caused by numerous other

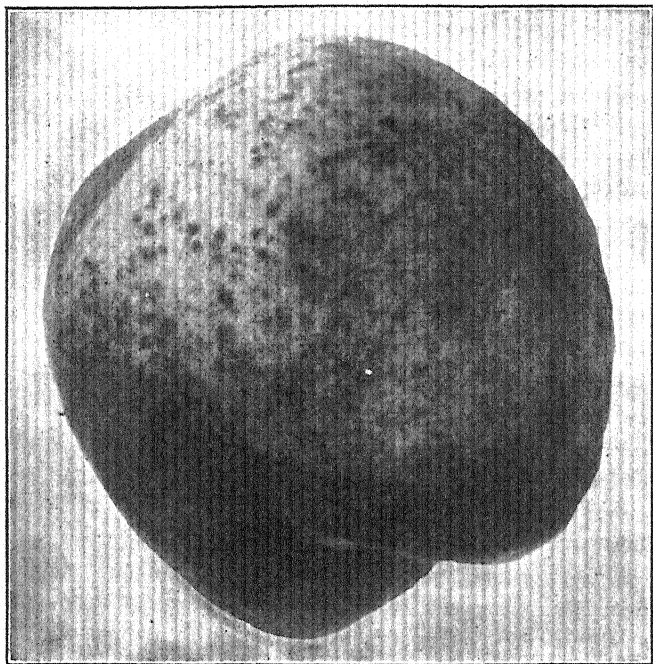


FIG. 210. — Mottling of peach fruit caused by yellows. (Photograph furnished by W. A. McCubbin, Pa. State Dept. Agr.)

things it is not a certain diagnostic character when taken alone. Sometimes only a single branch may show the symptoms of yellows while the remainder of the tree appears perfectly normal. Cutting out this branch, however, does not cure the tree.

*Diseases confused with yellows and little peach.* — There are several other agencies which may cause symptoms that are easily confused with the virus diseases of peach. The yellowing of foliage and sickly appearance associated with peach yellows is not confined to that disease alone and the other symptoms of yellows should be evident in addition to yellow foliage before a certain diagnosis is made. Some of the agencies causing symptoms likely to be confused with yellows are: (a) soil

deficiencies, (b) climatic factors such as winter injury, drouth, etc., (c) improper cultural practices, (d) injuries by borers or rodents, (e) other plant diseases such as root-rotting fungi, (f) mechanical injuries

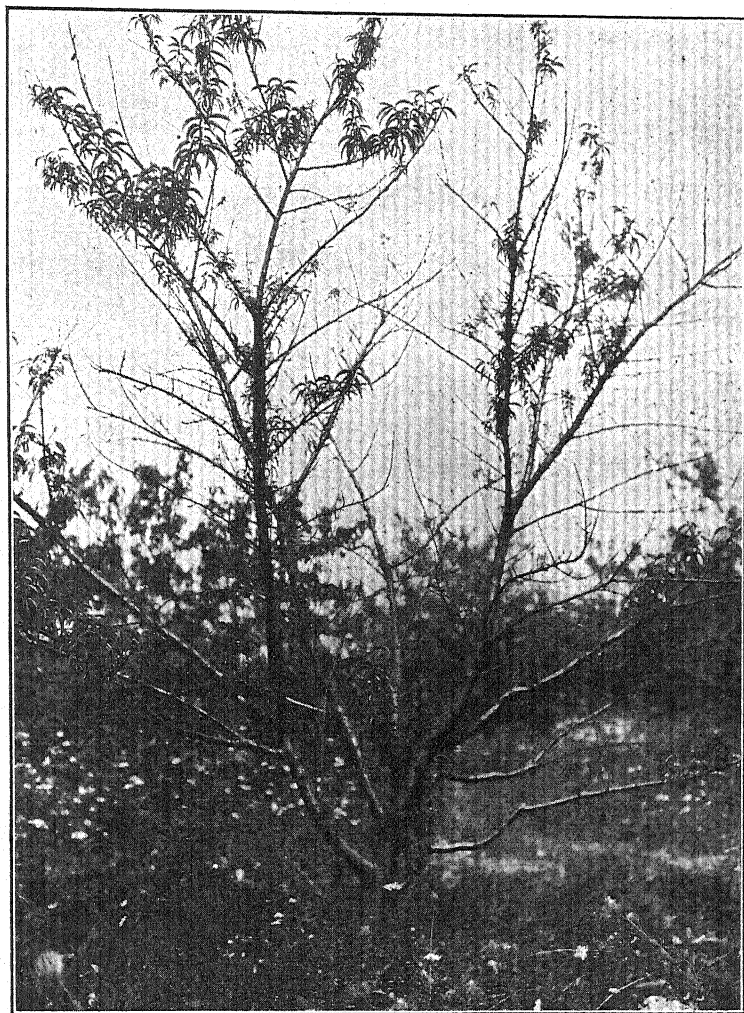


FIG. 211. — An old peach tree in the last stages of yellows. (Photograph furnished by W. A. McCubbin, Pa. State Dept. Agr.)

such as girdling by label wires (7). In order to be sure of making a correct diagnosis one should go through a process of elimination until all of these possible agencies have been disposed of before concluding that the trouble is certainly yellows.



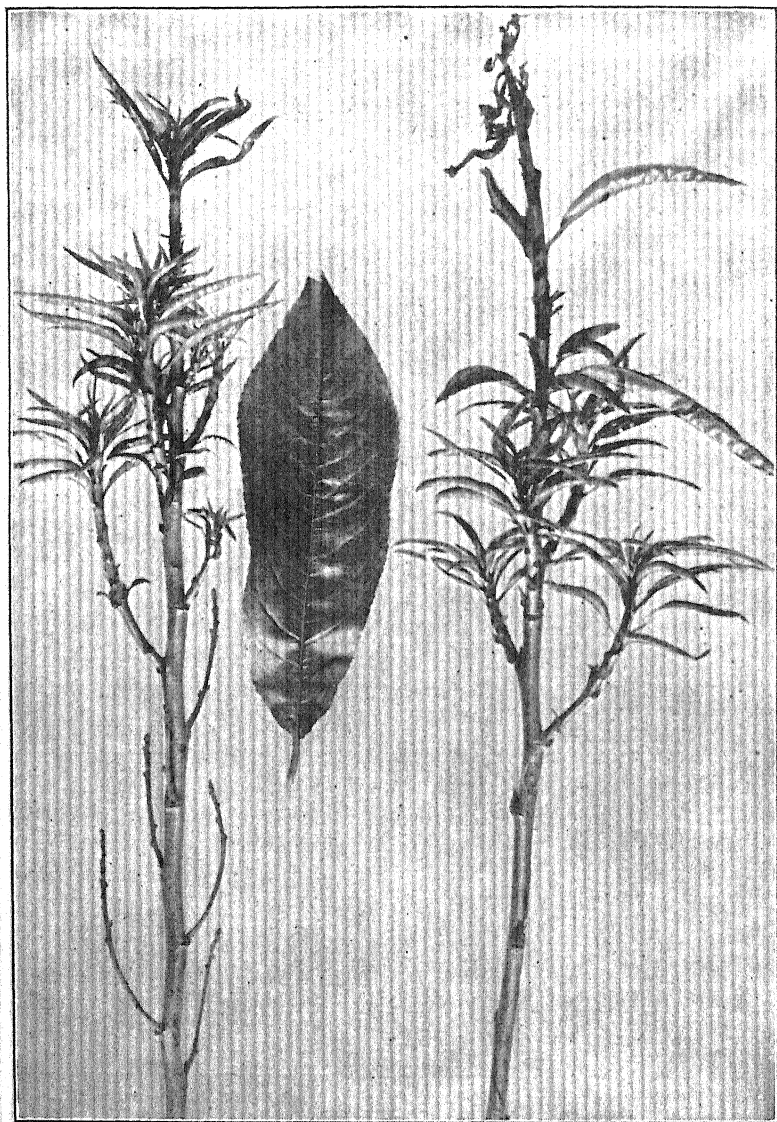


FIG. 212. — Two peach shoots showing symptoms of yellows. Healthy leaf in center. (Photograph furnished by W. A. McCubbin, Pa. State Dept. Agr.)

**Cause.** — As was the case with other virus diseases much speculation has been indulged in as to the cause of peach yellows and many theories have been advanced to account for it. It was at first thought by many that it was due to some unfavorable climatic or soil conditions. During the earlier investigations of the trouble many fertilizer experiments were tried on the theory that a correction of certain soil deficiencies might control the disease. All these failed. When the enzyme theory was advanced in connection with tobacco mosaic, some investigators suggested that the theory might also apply to peach yellows. When the communicability of the disease became apparent the usual attempts to associate various fungi, bacteria, and insects with the disease as causal agents were made, but in the end all these suggestions were discredited. Careful experimental work finally established beyond much doubt that the disease belongs to the general group now known as virus diseases, although perhaps differing in some details from such diseases as some of the potato mosaics. It should be borne in mind that since no successful sap inoculations have been made with the yellows "contagium" it has not been definitely proven that this causal agency is a filterable virus. In other respects, however, it meets the requirements and since no causal organism has been demonstrated for the disease it is logical to conclude that it is a virus disease.

**Transmission.** — Based on our knowledge of the virus diseases in general there are several possibilities which should be investigated in determining the methods of dissemination of peach yellows. These are: (a) insects; (b) budding and grafting; (c) seed; (d) pruning tools; (e) soil; (f) pollen; and (g) sap from diseased trees. There does not seem to be any authentic record of insect transmission of any of the virus diseases of the peach. This seems rather strange in view of the fact that insect transmission has been unquestionably proven for many of the other virus diseases. It is to be expected that future investigation will bring forth confirmation of this type of dissemination in view of the fact that there is every indication that the disease does spread in some manner from tree to tree in the field. In the past it has been claimed that the virus is transmitted through the seed from diseased trees to the seedlings. Recently published evidence from the New Jersey Station (4) indicates that there is no proof that the disease is carried in the seed. Very few seeds from yellows trees germinated and the few seedlings that were produced from diseased seed showed no evidence of the disease. Transmission of the disease through the soil has been suggested but there is no confirmation of this theory. In view of the fact that the transmission of spindle-tuber of potato by the cutting knife has been recently established, the possibility of trans-



mission of peach yellows by pruning tools should not be overlooked. To date, however, there is no proof that this occurs. Attempts to determine if the yellows virus is carried by pollen have thus far given negative results. A rather surprising development in the investigations of this disease is the fact that all attempts to transmit the disease by inoculating virus-carrying juice into healthy trees has failed.

*Buds and grafts.* — The one unfailing method of transmission thus far demonstrated is through budding and grafting diseased material onto healthy trees. This was suspected for a long time in actual nursery practice and has been confirmed abundantly by experimental work. Even if the bud fails to grow the disease may be transmitted anyway. It has been transmitted by inserting a piece of bark from a diseased tree into a healthy one. There seems to be a difference in the virulence with which buds from different diseased trees transmit the disease and in the time required for symptoms to appear after the budding has been done. Sometimes the disease develops unmistakable symptoms within a year and again the symptoms are not evident until several years after the diseased bud has been inserted in a healthy tree. Why this is so is not known.

*Control.* — In view of the fragmentary nature of our knowledge of this disease it is difficult to make complete and unequivocal recommendations for controlling it. There is one point, however, upon which we can be positive. This is based upon the fact that the disease is transmitted by budding and grafting. The next most important point, while not based upon positive knowledge as to the exact means, rests upon the observational evidence that the disease does spread in the field. With these two things in mind, we can make two positive recommendations, namely: (a) use buds and scions from trees absolutely free from yellows, and (b) destroy quickly and completely any tree known to be affected with yellows. The greatest care is necessary in selecting budding and grafting wood for nurseries. If diseased stock is used the entire output of a nursery may be infected with yellows. Thus the orchardist who plants such nursery stock is doomed to failure from the beginning. In spite of the fact that buds are positively known to carry the disease, statements in the literature indicate that there is little evidence of wholesale dissemination of the disease on nursery stock, but that in the majority of cases there seems to be a gradual spread in an irregular and ununiform manner from a single tree or a few diseased trees to others in the orchard. Although the means of dissemination in the field is not known and there are many inexplicable eccentricities in the way in which it does spread, yet the evidence of field spread is so great that it is safest to destroy all diseased trees immediately. The

urge to follow this practice is made stronger by the fact that after a year or two most of the diseased trees are worthless so there is no incentive to leave them in the orchard in the hope of obtaining a few good crops before they finally die.

There are other secondary recommendations that should be made on general principles, such as selecting well drained orchard sites; the use of vigorous, clean-appearing nursery trees; thorough cultivation and good care of the orchard; and the removal of all suspicious trees or trees of inferior quality whether definitely known to be contaminated with yellows or not.

### Little Peach

Little peach is another disease attacking this fruit which is closely related to yellows, but seems to be distinct from the latter. It has not been known nearly as long as yellows, being first discovered in Michigan in 1893, as far as available records go. It was reported from New Jersey in 1905. The native habitat of the little peach disease is not known but it may have come from Japan. It has not yet become as widely distributed in this country as peach yellows, but is found in some regions where yellows has long been prevalent. The reports of little peach come mainly from Michigan, New York, New Jersey, and Ontario, Canada. It occurs on certain varieties of plums as well as on peaches.

Little peach is distinguished from yellows mainly by the fact that the fruit is smaller than normal and ripens later than usual. The size of the fruit and also the lateness of ripening varies in different cases. The average delay in date of ripening is about ten days. There is also lacking the wiry twig growth so characteristic of yellows. The foliage exhibits characteristic symptoms but they are rather difficult to distinguish from symptoms of yellows in the foliage. The leaves of trees affected with little peach exhibit a lighter or yellowish green color and many of them are rolled or drooped, especially at the base and in the center of the tree. In order to recognize foliage symptoms of little peach it is necessary to be very familiar with the normal foliage characteristics of different varieties of peaches.

Little peach is apparently a virus disease similar to yellows since it also is transmitted by budding, and it apparently spreads in the field in the same manner that yellows does. Instances are on record in which both yellows and little peach occurred on the same tree. As far as the known facts concerning these two diseases are concerned, and considering the similarities in their behavior, it seems logical to recommend the same control measures for little peach as for yellows.

### Peach Rosette

The rosette disease has been known for a longer time than little peach. It was first noted in Georgia in 1881. By 1891 its known range had extended to Kansas and South Carolina. It is now known also in Tennessee, West Virginia, Missouri, Oklahoma and Alabama. It has been transmitted artificially from peach to plum, apricot, cherry and almond, by using diseased buds. Rosette is very destructive in certain regions, working even more rapidly than yellows.

The characteristic symptom of rosette is the bunching of the foliage at the tips of twigs and spurs. Usually a whorl or rosette of short sprouts develops due to the pushing out of more than the normal number of buds in the spring. The shoots from these buds make only a stunted growth. The foliage becomes yellow and falls prematurely. There is a tendency for the dormant buds to unfold in the summer and autumn. The fruit on diseased trees does not ripen before the normal time as in yellows, but it falls prematurely.

Sufficient experimental work has been done to demonstrate that rosette is caused by a virus as are yellows and little peach. Smith (13) conducted budding experiments in Georgia in 1890-91 which proved the infectious nature of this disease. In 1923 McClintock (8) published the results of some extensive cross inoculation experiments conducted at the Georgia Experiment Station. By means of the usual budding procedure he transmitted the disease from peach to plum, apricot and cherry; from apricot to almond; from plum to almond; from plum to peach; and from almond to peach. He failed to secure transmission of rosette through the soil or by means of sap from diseased trees. Numerous attempts to demonstrate the transmission of rosette by means of aphids, beetles and leafhoppers failed. The disease gives every evidence of being contagious in the field and the same control measures apply to rosette as to yellows and little peach.

#### REVIEW QUESTIONS

1. Describe the symptoms of peach yellows.
2. Describe the symptoms of little peach and peach rosette, and compare and contrast each with peach yellows.
3. What is the only method by which peach yellows has been artificially transmitted to date?
4. By what means has little peach been experimentally transmitted? Peach rosette?

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## LABORATORY STUDY OF VIRUS DISEASES

A. Symptoms. — Examine specimens showing the symptoms of any of the virus diseases available. Preserved specimens of some of the more striking diseases such as rugose mosaic of potato, cucurbit mosaic and curly-top of beet may be used but are not as satisfactory as fresh specimens. If season and conditions permit some of the virus diseases may be studied in the field. Specimens grown in the greenhouse under optimum conditions for the expression of the various symptoms are desirable, if it is possible to have such material for class study.

1. Learn to identify as many of the virus diseases of potatoes as are available for study. What are the distinguishing marks of mild mosaic? Rugose mosaic? Leaf-roll? Spindle-tuber? Of any other potato virus diseases available?

2. Note the symptoms of cucurbit mosaic on both foliage and fruits of the cucumber. Will this virus attack other members of the cucurbit family? Note the symptoms on the milkweed or any other susceptible plant. What other virus disease has been discovered on the squash, a near relative of the cucumber, in recent years?

3. Compare the symptoms of beet curly-top with those of the potato virus troubles and with the cucumber mosaic.

4. Describe the symptoms of the three peach virus diseases so as to distinguish them from each other, and from virus diseases of other plants.

B. The virus. — Since the virus cannot be seen, try to learn something about it by studying its behavior in various cases.

1. *Potato viruses*. — In all known cases, how is the virus carried over from season to season? In which cases can the disease be transmitted artificially by means of expressed juices and leaf-mutilation inoculations? In which cases by core-grafting? By the cutting knife? What insects transmit potato viruses? Which potato viruses are insect transmitted?

2. *Cucumber mosaic*. — How does this disease overwinter? How transmitted in nature? Artificially?

3. *Curly-top*. — How does beet curly-top differ from potato and cucumber virus diseases in manner of perpetuation and transmission?

4. *Peach viruses*. — Compare the peach virus diseases with each other and with the virus diseases of potato, cucumber and beet as to manner of perpetuation, dissemination and infection, in so far as these are known.

C. *Notes*. — Write notes on any of the virus diseases studied, describing symptoms and manner of perpetuation, dissemination and infection.

## CHAPTER XXV

### NON-PARASITIC DISEASES

There are a large number of non-parasitic troubles to which plants are subject. In the broadest sense these include all kinds of mechanical injuries or wounds, as well as those diseases which apparently arise within the plant itself and which have been considered as constitutional in nature. The latter group includes the diseases which are sometimes referred to as "physiological" diseases, and is the group from which examples will be chosen for further discussion. The troubles of this type are due in large part, if not entirely, to the influence of adverse environmental factors which not only interfere with the normal physiological functions of the plant, but usually bring about structural changes, also, which are exhibited as the characteristic symptoms of the disease. The environmental factors concerned may be considered under two heads: those that exert their influence in the field during the developmental period in the life of the plant, and those to which plant products such as fruits and vegetables are subject after harvest. Of course some of the "physiological" diseases are due in some degree to both field conditions and those existing subsequent to harvest. The factors involved in causing the non-parasitic diseases of this type which at least have their beginnings in the field are chiefly climatic and weather conditions. The soil is concerned to the extent that the type of soil has a great deal to do with the amount and uniform constancy of the water supply to the roots of plants. The factors which influence the development of non-parasitic troubles after harvest are the temperature, humidity, and aëration to which the products are subject immediately following harvest and later in storage or during transportation to market.

Of all the factors concerned in causing those physiological disturbances which are due primarily to field conditions during the growing season, that of the proper water balance is the most important. The water requirements of plants are delicately adjusted and any great disturbance in the amount and regularity of the supply is quickly reflected in the health of the plant. Plants take in water through the roots and give it off largely through the leaves. The outgo must not exceed the intake or the plants will suffer correspondingly. The rate of transpiration is determined by the temperature and humidity of the air. The rate

of water intake is influenced by the amount of water in the soil, the physical condition of the soil with reference to its water-holding capacity, the soil temperature, and probably other factors. On a hot day in summer when the relative humidity of the air is low an apple tree will transpire enormous quantities of water. If the water supply in the soil is adequate and the soil conditions are such that the plant can readily absorb it, the balance is not disturbed. On the other hand, if for some reason the plant can not get water as rapidly as it evaporates from the leaves, ill effects are bound to result. Even if the soil moisture and other conditions are satisfactory it sometimes happens that the root system of the plant itself is not in proper condition to supply the demand for water. Root systems develop poorly in shallow or hardpan soils and where waterlogged conditions prevail. Such defective root systems increase the probability of an inadequate water supply during weather when transpiration is excessive. Cultural practices which cut off a large percentage of the feeding roots and rootlets of the tree have the same effect. In other words, there must be the proper balance between root system and leaf expanse in order to maintain the water balance in the plant even though the water supply in the soil is adequate. In case of fruit trees under conditions where the evaporation exceeds the intake it has been shown that water is sometimes actually drawn out of the fruits and evaporated through the leaves. On the other hand, the sudden stopping of transpiration at night when the roots are absorbing water rapidly may sometimes result in gorging the plant with water and bursting or waterlogging the delicate fruit tissues. Just how this disturbance of the water balance causes the various physiological disorders is not very well understood but some of the theories will be mentioned under the individual diseases to be discussed later.

Most of the better known "physiological" diseases occur on fruits. The apple is subject to an exceptionally large number of these troubles, but other fruits such as the pear, prune and grape are frequently severely injured by one or more kinds of non-parasitic troubles. Naturally in a group of diseases apparently so closely related in nature, and about the cause of which there has been so much uncertainty and speculation, there should be a great deal of confusion as to the distinctness and identity of the different troubles. This has led to confusion and difference of opinion in the nomenclature. A partial list of more or less distinct troubles that are quite widely recognized on apple fruits includes bitter-pit, cork, drouth-spot, water-core, Jonathan spot, scald, soft-scald, and internal breakdown. To these should be added spray-injury, sunburn and freezing injury, which are of somewhat different nature from the above, but certainly non-parasitic troubles of common occurrence.

Potatoes are subject to certain non-parasitic troubles also, chief of which are black-heart and internal brown-spot. Blossom-end rot of tomatoes is apparently a disease of this type also. In the Pacific Northwest the prune is subject to a kind of drouth spot and corkiness presumably similar in cause to the corresponding diseases in apples. This list by no means includes all the non-parasitic diseases, but serves to illustrate different types of this group.

Of the diseases mentioned above, those which originate in the field are bitter-pit, cork, drouth-spot, and water-core of apple, and also blossom-end rot of tomato and internal brown-spot of potato. Those originating in storage or transportation are scald, soft scald and internal breakdown of apple, and black-heart of potato. Certain of these diseases may appear either before or after harvest and even with some which become evident only after harvest, it has been shown that pre-harvest conditions help to determine the susceptibility of the fruit to the disease. Jonathan spot is one of these diseases manifesting itself either before or after harvest and one the occurrence of which seems to be particularly dependent on various conditions pertaining to the maturity of the fruit at the time of harvesting.

### Bitter-pit and Related Troubles

Caused by lack of balance in the water supply

This disease has been frequently referred to in the literature of plant diseases for a quarter of a century, and the first mention of troubles apparently referable to this disease was made over a half century ago. The Germans designate the disease as "stippen," while in the British Provinces and in the United States, it has been variously named bitter-pit, fruit-pit, Baldwin-spot, dry-rot and brown-spot. The first name applied to the disease in the United States was "Baldwin-spot," because of its common occurrence on that variety of apple. It is by no means confined to the Baldwin variety, however, so that when the name "bitter-pit" was suggested for the disease it met with favor and the latter name is now most commonly used in referring to this disease. The name "bitter-pit" seems to be more appropriate in view of the characteristic pitting of the surface of the fruit and of the bitter taste frequently imparted.

**History and distribution.** — The disease probably has existed from the time apples first came under cultivation but like many other diseases it was only after this fruit came to be grown commercially



on a large scale that the disease began to attract attention and to appear as the subject of horticultural and pathological writings. Jaeger (18) seems to have been the first to mention this disease. In 1869 he described a pitting in certain varieties of apples, which he supposed to be due to rapid transpiration. Wortman (28) named the disease "stippen" in 1892. In 1891 L. R. Jones (19) described an apple disease in Vermont under the name of "Baldwin-spot," which was undoubtedly the disease now known as bitter-pit. The trouble was first reported in Australia in 1886, in Canada in 1896, in South Africa in 1901 and in England in 1905. Cobb (5), writing in the *Agricultural Gazette* of New South Wales in 1895, first suggested the name "bitter-pit" for this malady. In 1908 Brooks applied the term "fruit-pit" to the trouble. Some of the most recent work on the cause and control of bitter-pit was done by Brooks and Fisher (2) in the northwestern part of the United States. At the present time this disease is known all over the world wherever the apple is grown extensively.

**Hosts and varietal susceptibility.** — Bitter-pit is best known on the apple but is not confined to this fruit. Pears and quinces are also susceptible. While it is not known that any variety of apple is entirely immune to bitter-pit under all conditions, yet there is a marked difference in varietal susceptibility. This fact has always been so evident that some of the earlier writers, in discussing the cause of the trouble, even suggested that the disease might be inherent in certain varieties. In America it was at one time supposed by some that the disease was peculiar to the Baldwin variety. The name "Baldwin-spot" first used to designate the disease in this country testifies to this fact. Other varieties which are commonly affected in America are Northern Spy, Rhode Island Greening, and Tompkins King. In Australia McAlpine (23) has listed a large number of varieties in order of their susceptibility. This order is not always the same in the different provinces of the Commonwealth. The following grouping was given by McAlpine for the State of Victoria in southwestern Australia.

*Very susceptible.* — Annie Elizabeth, Buncombe, Cleopatra, Cox's Orange Pippin, Lord Wolseley, Magg's Seedling, Northern Spy, Prince Bismark, Ribston Pippin, and Shockley.

*Medium.* — Delicious, Duchess of Oldenburg, Esopus Spitzenberg, Hoover, Nickajack, Perfection (Shepherd's), Prince Alfred, Rokewood, Sturmer Pippin.

*Slightly susceptible.* — Ben Davis, Dumelow's Seedling, Five Crown of London Pippin, Gravenstein, Jonathan, Munroe's Favorite, Pomme de Neige, Reinette de Canada, Rome Beauty, Rymér, Scarlet Nonpareil, Statesman, Stone Pippin, Winter Majestín.

**Symptoms.** — The symptoms of this disease are both external and internal. In well marked cases the external evidences of the disease are a distinct pitting and spotting of the skin (Fig. 213). The spots are more or less sunken as if a small rod having a blunt rounded end had been pressed endwise against the apple until a slight depression had been made. These pits vary in depth from a scarcely perceptible depression up to a millimeter or two in depth. The diameter of the spots also varies from one or two up to five or six millimeters. They

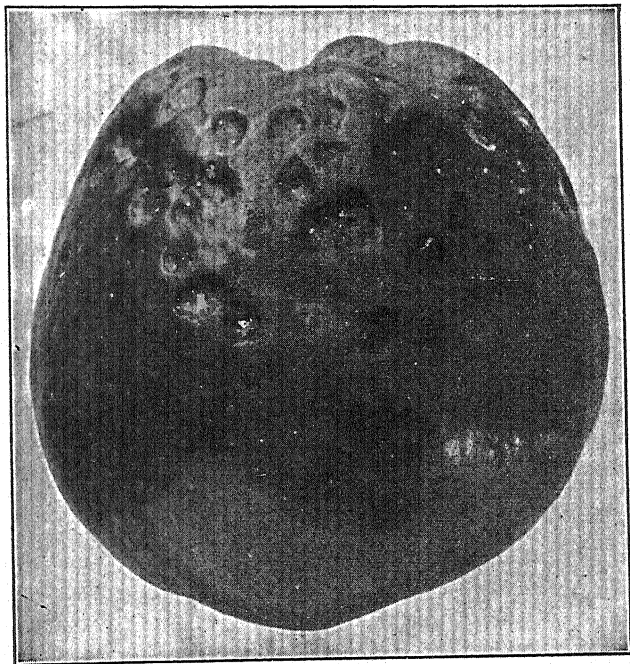


FIG. 213. — Bitter-pit of apple. (After Jackson, Ore. Agr. Exp. Sta. Crop Pest Rept., 1911-12.)

are usually more or less circular in outline. The color is variable. In well defined, advanced stages the spots are decidedly brown and dead in appearance. In earlier stages they are water-soaked, pale green, or a dark green in color. The distribution of the spots over the surface of the fruit is also quite variable. The depressions occur mostly on the upper or blossom-end half of the fruit, but may extend nearly to the pedicel. However, there is always an area with a radius of an inch or more around the stem end which is free from pits. The pits may be confined to one side of the fruit or they may extend all around it.

If the fruit is sectioned through the pits there will be seen a dome- or cone-shaped region of dead brown cells immediately beneath the surface. These areas of dead corky cells, which begin at the surface, extend down into the flesh a variable distance but generally to an average depth about equal to the surface diameter of the spot. Frequently, according to McAlpine, these dead brown spots will be found scattered through the flesh at greater depths with no surface indications, some even occurring near the core of the fruit (Fig. 214). Fruits may be found with all the spots showing at the surface, or again there are cases where no spots are in evidence on the surface of the fruit, but on cutting the fruit in two the brown spots are found scattered deep in the flesh. The latter condition has been termed "cork" by some investigators, as distinguished from bitter-pit proper. Several modifications of these symptoms have been described under various names. McAlpine described a form which he called "crinkle" in which the fruit is very much deformed and more or less dwarfed. He considered it a variation or phase of the general bitter-pit trouble. Mix (24) described "cork" and "drouth spot" as distinct types of non-parasitic apple diseases. If sections of the dead tissue are examined under the microscope the cells are seen to be collapsed and even in ripened fruit the dead cells are filled with starch grains whereas the healthy cells have no starch grains in them when ripe. According to McAlpine's first theory, the dead cells are collapsed, not burst, and the brown color is imparted by a mucilaginous substance of a pectic character. Later he concluded that the cells are burst due to high sap pressure.

**Cork.** — Although McAlpine apparently looked upon this type of injury as a variation of bitter-pit, there is good reason to consider it a distinct type of disease. Mix (24) has given it the most careful study and distinguishes clearly between bitter-pit and cork. The symptoms consist of internal, brown corky spots scattered through the flesh of the fruit (Fig. 214). In a sectional view these brown spots seem to be isolated and scattered indiscriminately through the fruit extending in nearly to the core. In reality the spots follow closely the course of the vascular bundles. The brown spots never show through the skin but the surface of the fruit may be thrown into a series of elevations and depressions. In the earliest stage of the disease, which may show up when the fruits are not much more than two centimeters in diameter, there are no external symptoms. Even when the fruit is mature an inexperienced person would sometimes have difficulty in diagnosing the disease from the surface evidences. Mix would use the term "bitter-pit" to designate only that type of trouble which occurs at or near the surface of the fruit.

**Drouth spot.** — Mix (24), and Brooks and Fisher (2) have described "drouth-spot" as a distinct form of this group of diseases. The symptoms may appear at any stage in the development of the fruit, but are more likely to become evident after the fruit is one-third grown. This trouble is typically shallow, ordinarily extending only a few cell-layers deep, but occasionally deep-seated lesions are found in which dead cells occur in wedge-shaped areas extending down toward the vascular

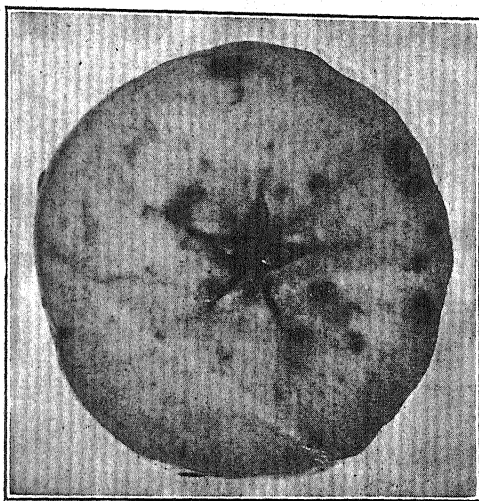


FIG. 214. — Cork spot in apple. (Photograph from files of the Ore. Agr. Exp. Sta.)

system. The first indication is a large irregular water-soaked area usually covered with drops of ooze. Upon cutting through these spots a thin layer of dead brown tissue is found. Later the skin of the fruit may become roughened and cracked. In other cases the skin over the spot may regain its normal appearance but the surface of the fruit will be depressed at this point giving rise to a misshapen apple.

**Cause.** — Many theories have been advanced to account for the occurrence of bitter-pit and while we probably now know the general factors concerned, the exact manner in which the injury is brought about is still a matter of conjecture. In the first studies conducted on the disease there was a natural tendency to look for some sort of parasitic plant or animal life as the causal agent. In 1892 Cobb (4) suggested the possible causal relationship of fungi and insects, and later again stated that bitter-pit is probably a kind of stigmonose. Other investigators also at first suspected parasites of some kind, but after a time, when diligent search failed to justify belief in this kind of a causal agent, they turned their attention to other possible causes. When spraying

with lead arsenate for insect control came into such common use, some were inclined to suspect that bitter-pit might be a form of arsenic poisoning. Further investigation, however, soon convinced many that the trouble was of a different nature and the term "physiological disease" came into quite general use in referring to this malady. Various theories along this line of thought now began to be formulated. In 1903 Clinton (3) stated that the disease probably resulted from too great loss of water. As early as 1869 Jaeger (18) had suggested that it was due to rapid transpiration. Massee (22) supposed that too high temperatures during the first period of ripening might cause the trouble. Diakonoff (10) thought the trouble was caused by too rapid growth of individual groups of cells in the flesh of the fruit. Griffiths (15) stated that bitter-pit is a constitutional weakness directly due to overgrowth. In 1909 Evans (11) propounded the bursting cell theory in which he states that rapid transpiration by day followed by sudden checking in evaporation at night-fall while the roots are still active results in an excessive sap pressure which bursts groups of cells here and there, giving rise to the dead, brown spots in the fruit tissue. The various causes to which bitter-pit has been attributed may be summarized as follows: (a) mechanical agencies; (b) unfavorable conditions of soil and weather; (c) insects; (d) fungi and bacteria. Of these only the second appears to have probability.

If we accept the prevailing opinion that a disturbance of the water balance in the plant is responsible for the trouble, the exact nature of this disturbance and the manner in which it acts still remain to be explained. Compiling and summarizing the more recent theories which have arisen out of this general concept, we have about four different hypotheses that have been propounded to account for the occurrence of bitter-pit. These are: (a) the sap concentration theory; (b) the starvation theory; (c) the burst-cell theory; and (d) the crushed cell theory.

The first theory is based upon the supposition that rapid transpiration, that is, in excess of absorption, results in a concentration of chemicals in the cell sap, especially plant acids, in amounts sufficient to cause the death of groups of cells.

The starvation theory postulates that severe drouth cuts down the amount of water entering the plant and at the same time interferes with the intake of sufficient minerals from the soil and results in the starvation of some cells due to lack of the necessary mineral elements.

Adherents of the burst-cell theory claim that the sudden checking of transpiration at night-fall, or for any other reason, while the roots are still actively absorbing large quantities of water, results in a high sap pressure sufficient to burst groups of cells at certain places in the fruit.

McAlpine and others have suggested that the small vasculars or vascular elements are also burst in the pitted spots.

The most recently advanced theory is the crushed cell theory propounded by Herbert (17). He uses as the basis for his argument the often observed fact that the groups of dead cells are always full of starch grains. He states that during the time when the starch is being transformed into sugar some cells will be free from starch while others are still gorged with starch grains. When a sudden rush of sap into the apple occurs, the cells in which the starch has been converted to sugar will naturally swell greatly. The force of this expansion may be sufficient to crush the groups of cells which are still filled with starch and which consequently do not swell so much nor so rapidly as the others because of their lower osmotic pressure.

It must be admitted that as yet none of these theories have become definitely established facts. While this is unfortunately true, yet certain facts have been established to give us a fairly definite idea of some of the conditions under which bitter-pit is likely to occur. Brooks and Fisher (2) have carried out extensive irrigation experiments in which the water supply was controlled so as to furnish some indication of the part played by water. Briefly stated their experiments warrant the following conclusions. (a) Heavy irrigation throughout the season greatly increases bitter-pit. (b) Medium irrigation followed by heavy irrigation causes even more pit than heavy irrigation alone. (c) Light irrigation decreases the disease. (d) Heavy irrigation early in the season followed by light irrigation toward the end of the growing season gives the least bitter-pit of any treatments tried. Bitter-pit may not become evident on the fruit until after harvest but the conditions under which the fruit is grown determine whether or not it is predisposed to the disease.

**Control.** — In the light of all that is at present known about this disease it would seem that it can be controlled to some extent under irrigation, but that little can be done where the natural rainfall is depended upon to supply the needs of plant growth. Field observations indicate that if, after a dry season, heavy rainfall occurs shortly before the apples are harvested bitter-pit is apt to develop in susceptible varieties. Practically all that can be done is to conserve the soil moisture as much as possible by cultural methods and thus provide the plant with as adequate and uniform a supply of water as possible. This practice will apply especially to cork and drouth-spot as well as to bitter-pit.

**Note.** — Laboratory exercise on bitter-pit and other non-parasitic diseases of the apple will be found following the treatment of apple-scald.

## REVIEW QUESTIONS ON BITTER-PIT

1. Describe the symptoms of bitter-pit.
2. Mention and discuss four different theories that have been advanced to explain the cause of bitter-pit.
3. Discuss the work done by Brooks and Fisher on the effects of varying the water supply under irrigation on the occurrence of bitter-pit. (Reference 2.)
4. In regions where the natural rainfall is depended upon, what can be done to combat this disorder?
5. Compare cork and drouth-spot in apples with the bitter-pit trouble as to both symptoms and cause. Can the presence of cork be detected without cutting the apple? (Reference 23.)
6. Compare bitter-pit, cork and drouth-spot with other non-parasitic diseases of apples such as water-core, scald, soft scald and Jonathan spot which are discussed in the following pages.

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### Water-core of Apple

#### Caused by disturbed water relations

Water-core is an apple disease of common occurrence in practically all apple-growing sections of the world. In the United States it is most serious in the irrigated districts of arid regions, although it is apt to occur anywhere with variable severity from year to year. The characteristic symptom is the watery or glassy appearance of the flesh of the apple. The location and extent of the glassy areas vary. Usually the disease is confined to the region in the neighborhood of the vascular bundles or immediately surrounding the core. It first appears as small dots around the vascular bundles. The water-cored areas increase in size until a larger or smaller portion of the flesh is affected. Sometimes nearly the whole apple is involved. In this case the watery condition may be apparent through the skin. Ordinarily there is no surface indication and the disease is not evident until the fruit is cut open. Water-core usually occurs in mature apples, although in some cases it may appear several weeks before maturity. The glassy or watery appearance is due to the fact that water or exuded cell-sap fills the intercellular spaces instead of air as is the case in the normal tissue. Just what causes this exudation of water into the intercellular spaces has been the subject of as much speculation and theorizing as has the cause of bitter-pit. One of the most commonly used explanations relates



the trouble to fluctuations in transpiration. When transpiration is suddenly checked the excess accumulation of sap in the cells fills them to overflowing and some water is forced out into the intercellular spaces. This theory coincides with one of the theories advanced to account for bitter-pit except that instead of the cells bursting under pressure the excess water exudes into the spaces without bursting the cells. Whatever the true explanation of this phenomenon is, certain facts concerning the contributing factors have been fairly well established through experimentation. According to Brooks and Fisher (1) the following conclusions are justified. (a) Apples from heavily irrigated trees develop less water-core than those from lightly irrigated trees. (b) Light irrigation followed by heavy irrigation causes less water-core than heavy irrigation followed by light irrigation. (c) Treatment of trees with nitrate or potash fertilizers decreases the amount of water-core as compared with untreated trees. (d) Apples that are shaded develop very little water-core while those exposed to sunlight show a strong tendency to water-core. (e) Water-core develops very extensively in sunburned apples. (f) Over-mature fruit shows a much greater tendency to become water-cored. (g) Higher sap concentration is correlated with greater tendency toward water-core. (h) Sap concentration increases with the maturity of the apple. (i) Sunburned apples have a very high sap concentration. (j) Heavy irrigation reduces both sap concentration and water-core. (k) In the present state of knowledge the most practical preventive known for water-core is picking the fruit at the proper stage of maturity.

#### REVIEW QUESTIONS ON WATER-CORE OF APPLE

1. Describe the symptoms of this disease.
2. Can the presence of water-core be detected without cutting the apple?
3. What explanations have been offered as to the cause of water-core?
4. According to Brooks and Fisher what factors contribute toward the production of water-core in apples? (Reference 1.)

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### Apple-scald

Caused by certain gaseous by-products of metabolism

This is a non-parasitic disease of the apple which appears only in storage or on the market. It usually becomes more evident a few days after the fruit has been removed from storage and placed on the market. Sometimes there is no indication of the trouble until the apples have been out of storage several days when a high percentage of fruits will suddenly develop the scald. The disease is of great economic importance, not so much because of the absolute immediate destruction wrought, but because of the decrease in salability and market value due to the unsightly appearance of scalded apples. The fact remains, however, that scalded fruit deteriorates rapidly and soon becomes worthless, especially if invaded by secondary rot fungi.

**Symptoms.** — Apple-scald symptoms begin with a superficial brown tinting or discoloration of the skin of the fruit. Usually it involves only

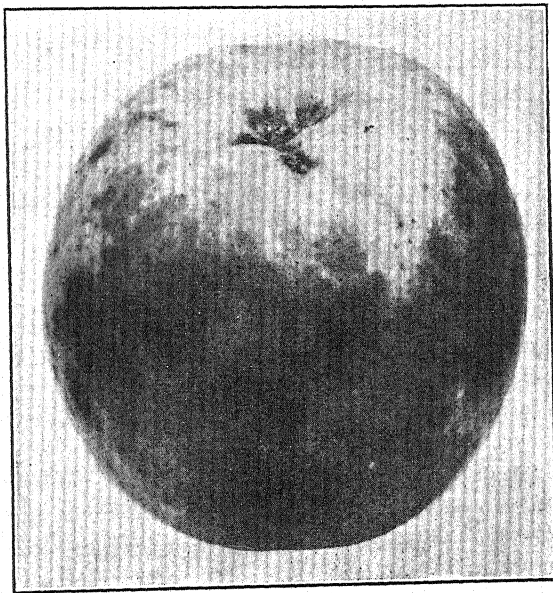


FIG. 215. — Scald, a storage trouble of apples. (Photograph furnished by D. F. Fisher, U. S. Dept. Agr.)

the outer cell-layers, but ultimately the flesh may become dead and brown to the depth of half an inch or more. In later stages it resembles apple rot more or less, but scald spreads out in irregular shaped areas (Fig. 215) and is generally uniformly shallow while the fungous rots are

usually in definite circular spots and the rotted tissue extends into the flesh in a cone-shaped mass with the apex of the cone toward the core of the apple. In severe cases, the skin of the apple breaks down and sloughs off. Scald is peculiar in that it is more prevalent on the greener side of the apple. Highly colored fruit surfaces are more resistant to scald.

**Cause.** — The fact that scald usually appears after the fruit has been out of cold storage for a few days has led to the supposition that the change from a cold to a warmer temperature causes the disease. This is not true, but the change of temperature does bring about certain changes which make evident the injury which has already resulted from other causes, but has not yet become conspicuously visible. The real cause of apple-scald is said to be certain esters which accumulate in the tissues and in the surrounding air. There are several other contributing causes or factors which render the fruit more susceptible to scald. These contributing factors are of two general classes: (a) the orchard conditions under which the fruit grows up to harvesting time, and (b) the packing-house, transportation and storage conditions (6).

*Effect of orchard conditions.* — Experiments have shown that certain conditions existing in the field during the growth of the fruit and at the time of harvesting have an effect upon the amount of scald that may develop later when the fruit is removed from storage. These are the degree of maturity at picking time, the size of the fruit, and the soil moisture in the orchard. Various tests have shown that fruit picked in an immature condition is more susceptible to scald than well matured fruit when stored under the same conditions. Too heavily irrigated orchards are likely to produce fruit which is more susceptible to scald than less heavily irrigated orchards.

*Conditions in packing-house, storage and transportation.* — Several factors enter into the question of susceptibility to scald after harvesting. Some of these are temperature, delayed storage, aëration and ventilation, kind of packages and humidity. In relation to temperature scald is like the fungous rots in that high temperatures are conducive to its development while low temperatures tend to hold it in check. Apples that are stored immediately at cold storage temperature are much less liable to develop scald later than fruit held at outside temperatures several days before placing in cold storage. Aëration and ventilation have to do with both the storage room and the package or container. A decided difference has been noted between the amount of scald developing in the middle of the stack of boxed fruit and that occurring near the aisle where there is more ventilation. Much less scald developed in containers on the aisle. Apples stored in tight barrels show

more scald than those stored in slatted boxes or baskets. If openings for ventilation are made in the barrels less scald develops.

*By-products of fruit metabolism.* — It has been shown beyond much doubt that the actual killing of the fruit tissues which results in the appearance of scald symptoms, is accomplished by certain gaseous by-products of the nature of esters. Scald has been produced experimentally by exposing apples to the vapors of ethyl acetate, amyl acetate, and methyl butyrate. Proper ventilation allows the injurious gaseous by-products, whatever they are, to be carried away by air currents before they can injure the tissues of the fruits. The contributing causes discussed above are simply factors or conditions which are favorable for the production and action of these deleterious gases.

*Control.* — The control of apple-scald necessarily centers around the removal of the conditions favorable for the production and action of the esters which cause the trouble, and also the removal of the harmful gases themselves after they are formed. This involves harvesting the fruit at the proper stage of maturity and immediate storage at sufficiently low temperature. The removal of the gases as rapidly as formed can be accomplished in two ways, namely, by proper ventilation, and by absorption by certain oils. It has been discovered that some of the colorless and odorless mineral oils will absorb the gases causing scald. The most effective way of using these oils yet found is in the form of oiled paper wraps. The use of these oiled wraps has been found commercially economical and feasible, and they reduce the scald to practically nothing. The use of shredded oiled paper in barrels has been found as effective as wrapping each apple individually, if an abundance of paper is used, so that all apples come in contact with the oiled paper.

#### REVIEW QUESTIONS ON APPLE-SCALD

1. Describe the symptoms of apple-scald so as to distinguish it from other non-parasitic diseases of apple fruits.
2. At what stage in the life of the apple does scald appear?
3. What effect do orchard conditions have upon the development of scald?
4. What conditions of storage are conducive to the production of scald?
5. What is the immediate cause of scald?
6. By what two methods may the deleterious esters be removed before damage results?

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#### LABORATORY STUDY OF NON-PARASITIC DISEASES OF APPLES

**A. Symptoms.** — Examine apples showing symptoms of the several non-parasitic disorders, such as bitter-pit, cork, drouth-spot, water-core, scald, Jonathan-spot, and soft-scald. In each case observe both the surface symptoms and those which are evident in a sectional view of the fruit. Which ones show both surface and internal symptoms? Which are only skin deep? Which ones can be detected only after the fruit has been sectioned? How can Jonathan-spot be distinguished from bitter-pit? How do the symptoms of soft-scald differ from those of common scald? In all cases where the flesh is affected note the color and texture of the affected areas. In the bitter-pit, cork, drouth-spot and water-core specimens, examine the tissues of the affected areas carefully to try to see if the vascular elements are involved.

**B. The cause.** — Make a careful search through the text and the references for all available information on the cause of each of these troubles.

**C. Notes.** — Write notes covering the symptoms of each disease; a brief statement of what is known of the cause in each case; and the best recommendations available for the prevention of these various disorders.

### Blackheart of Potatoes

Caused by high temperatures and poor ventilation

The attention of plant pathologists was called to this trouble during the years 1910, 1911, and 1912 by shippers and dealers in potatoes. The disease was of common occurrence in car-load shipments and was suspected to be due to either chilling or overheating. The disease is characterized by the occurrence of darkened or black areas in the center of the tuber as the name "blackheart" suggests (Fig. 216). The darkened area varies in size and shape. Sometimes it is small, almost round and regular in outline. Again it may occupy half or more of the interior of the tuber. Irregular projections may radiate from the spot. It may be a solid black area or may consist of a blackened ring surround-

ing a gray center. After several days the blackened area shrinks leaving a hollow space in the center surrounded by a black border of shrunk tissue. Ordinarily there are no surface indications of the trouble and it is discovered only upon cutting the tuber open

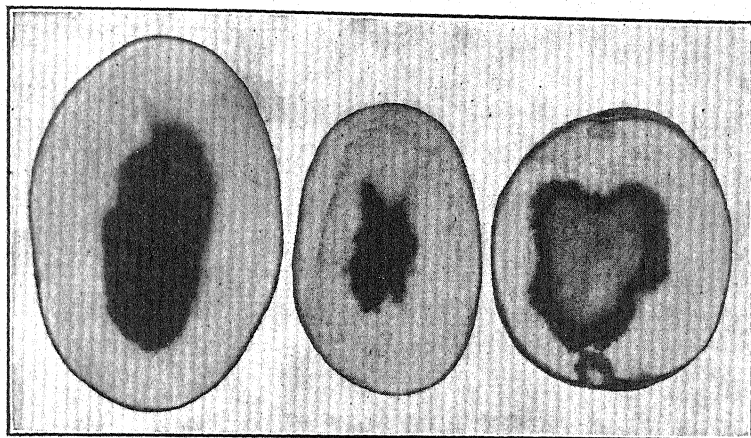


FIG. 216. — Cross sections of potato tubers showing typical symptoms of blackheart. (After McKay, Ore. Agr. Exp. Sta. Cir. 24.)

In 1913 Bartholomew (1) produced the disease experimentally by subjecting tubers to rather high temperatures. He found that he could cause the blackheart condition by exposing tubers to a temperature of 100° to 113° F. in a drying oven for from 18 to 48 hours, depending on the size and variety of the potatoes. At this time he stated that apparently heat was the chief, if not the only, factor concerned. In a later paper (2) he stated that a lack of oxygen was probably also a factor. It is thus evident that this disease is largely if not entirely a respiratory trouble.

Later Stewart and Mix (4) published additional data on the factors involved in causing blackheart. They discovered that blackheart could be produced at much lower temperatures than those reported by Bartholomew if the air were excluded from the tubers. They sealed tubers in jars and tanks and found that with a volume of air equal to the volume of the tubers, blackheart was produced at a temperature of 70° F. after a confinement of ten or twelve days. At a temperature of 55° to 60° F. about twenty days were required to produce the symptoms, and at 40° F. it required from twenty-three to forty days. Thus it is evident that the occurrence of blackheart is not dependent upon a single combination of factors but may be produced by various combinations of the three factors, temperature, deficient oxygen supply, and the length of

time during which the tubers are exposed to these conditions. In 1924 Bennett and Bartholomew (3) confirmed these findings and added further data. It follows from these facts that blackheart may occur in cool storage quarters as well as under conditions of overheating, provided there is not sufficient aëration. Control measures, then, hinge upon the avoidance of too high temperatures and the provision of proper ventilation.

#### LABORATORY STUDY OF BLACKHEART

**A. Symptoms.**—Examine potato tubers affected with blackheart. Can the trouble be detected without cutting the tuber? Are the symptoms as marked when the tubers are first cut open as after the inner tissues have been exposed to the air for some time?

**B. Cause.**—What causes blackheart? How was the cause determined? Look up the references on blackheart and note what apparatus was used and the process followed to produce this trouble experimentally. If facilities and time permit try some of these experiments in the laboratory.

#### REVIEW QUESTIONS ON BLACKHEART

1. Describe the symptoms of blackheart in potato tubers.
2. When and under what circumstances was this trouble brought to the attention of plant pathologists?
3. What factors were demonstrated to be instrumental in causing this disease? (References 1, 2, 3, 4.)
4. Discuss the various combinations of these factors which may result in blackheart. (References 3 and 4.)

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#### Miscellaneous Non-parasitic Diseases

It would be manifestly impossible to enter into a detailed discussion of all the non-parasitic troubles in a text of this sort but there are so many of them which are of more or less economic importance that brief mention will be made of some of these diseases here and a list of references appended wherein the reader can secure additional information upon any of them in which he may be especially interested.



**Jonathan-spot.** — This is a skin-deep spotting of the apple fruit. It is especially prevalent on the Jonathan variety, hence its name, but may occur on other varieties. It may appear either before or after harvest. The typical spots are brown or almost black, slightly depressed, but affecting only the skin, and are one-eighth to one-fourth inch in diameter (Fig. 217). Late picking and delayed storage increase

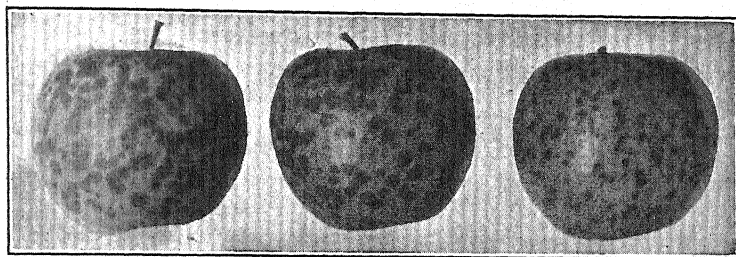


Fig. 217. — Jonathan-spot on apple. (Courtesy Pomology Section, Iowa Agr. Exp. Sta.)

the amount of Jonathan-spot. Low storage temperatures are unfavorable for the development of this trouble. Recent investigations (27) indicate that the acidity in the skin of apples is less in the spotted areas than in the other regions. This is thought to account for the color of Jonathan-spot. It is suggested that the acidity may be maintained by the use of paper wrappers impregnated with a harmless acid. (References 5, 6, 7, 11, 19, 22, 23, 27.)

**Soft-scald.** — This is a superficial browning of the skin of apples. It occurs in more definite areas with more sharply marked limits than

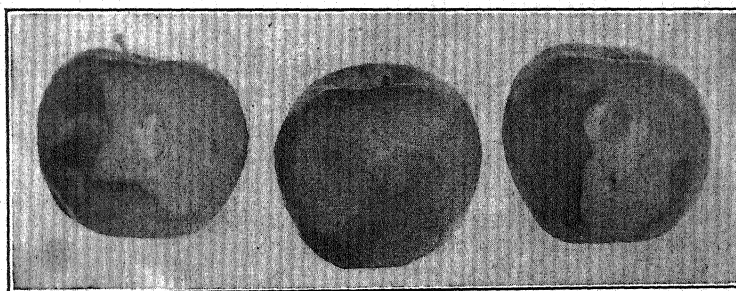


Fig. 218. — Soft-scald on apple, a storage trouble differing somewhat from common scald. See Fig. 215. (Courtesy Pomology Section, Iowa Agr. Exp. Sta.)

the ordinary scald previously described. At first the trouble is only skin deep but later extends some distance into the flesh. The affected portion finally becomes sunken and somewhat corky (Fig. 218). The



degree of discoloration varies from a light shade of brown to a much darker color. The size of the soft-scald areas varies from small spots to almost the entire surface of the fruit. The time of picking and the time of storing are important factors in determining the amount of soft-scald developing in fruit. Delaying storage for three weeks after picking gives the least soft-scald. This is the opposite of the results obtained with Jonathan-spot. (See Reference 9 under Apple-scald.)

**Blossom-end rot of tomatoes.** — Tomatoes are often subject to a type of dry-rot occurring at the blossom-end of the fruit (Fig. 219) which may be confused with fungous and bacterial rots which are also

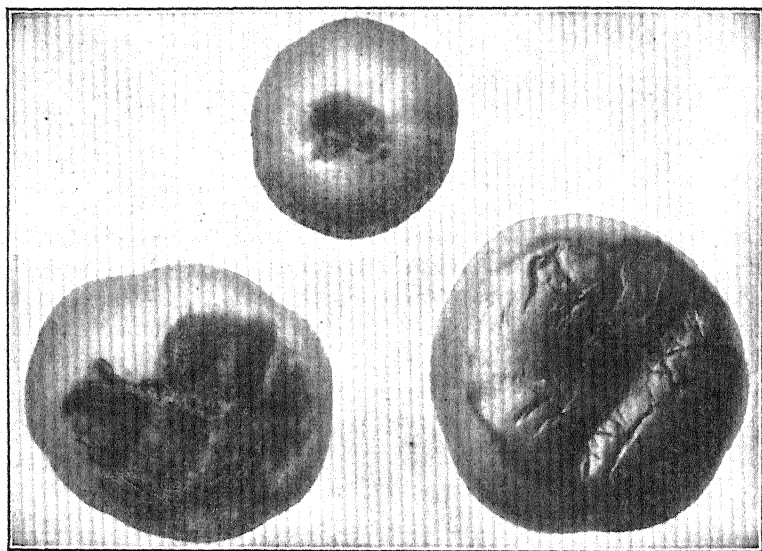


FIG. 219. — Blossom-end rot of tomato. (After McKay, Ore. Agr. Exp. Sta. Crop Pest Rept., 1915-20. 1921.)

common diseases of the tomato. This disease, however, is apparently not associated with organisms as a primary cause. It always occurs at the point or blossom-end of the tomato and is apparently caused by excessive transpiration or a shortage in the water supply. It occurs in the field under drouth conditions and is common in the greenhouse, even, where the plants are irrigated. Its frequent occurrence under glass where presumably the water supply can be regulated is probably due to the fact that the soil in greenhouse beds is usually shallow and consequently it is more difficult to maintain a uniform supply of water around the roots of the plants and extreme fluctuations in the soil water seem to favor the development of the disease. (References 14, 15, 24.)

**Internal brown spot of potato tubers.** — This disease in potatoes resembles cork in apples and is probably due to similar conditions. Brown corky spots of irregular shape and size are found scattered at random through the flesh of the tuber. No superficial symptoms are evident and the disease is observed only when the tubers are cut open (Fig. 220). The exact cause of the trouble is not known, but it is supposed to be due to lack of soil moisture. The type of soil is also probably a factor in so far as it influences the amount and regularity of the water supply.

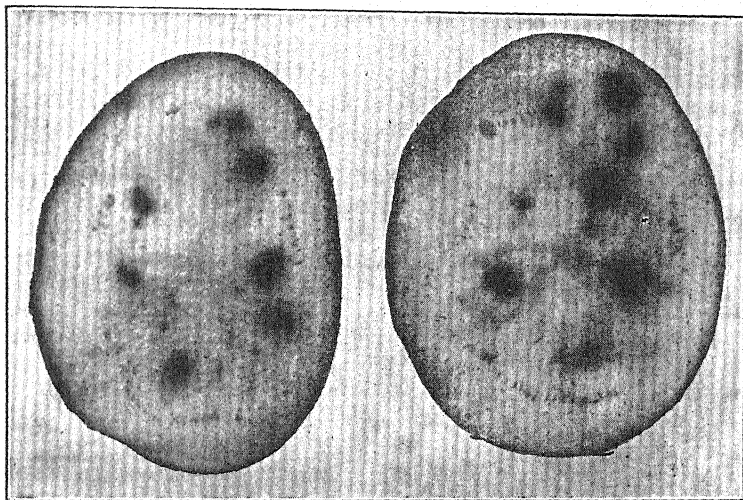


FIG. 220. Sections of potato tuber showing internal brown spot. (Photograph by Bailey, Ore. Agr. Exp. Sta.)

**Apple rosette.** — This disease is characterized by dense tufts of leaves at the tip of twigs. The condition is brought about by the failure of the twig to elongate normally and consequently all the leaves that would ordinarily be distributed at uniform distances along a twig one or two feet long are clustered on a dwarfed shoot only an inch or two in length. The terminals are affected first, but rosettes may appear later on short side branches. The leaves of the rosette do not develop normally, but are narrower and more slender than healthy leaves. The apple rosette disease resembles the peach rosette in some respects, but it apparently is not a virus disease as is the latter. All experimental work to date indicates that it is a functional or nutritional disease. Great improvement in rosetted trees has followed the growing of a legume cover crop in the orchard for three or four years, and trees have been known to recover completely after five years of this treatment. (References 18, 21.)

**Gum spot and cork of prunes.** — In the prune-growing sections of the Pacific Coast states the long, dry summers cause serious drouth effects in prunes. A symptom very frequently encountered consists of a dead, slightly-sunken spot on the surface of the prune much resembling bitter-pit in apples, except that the dark color of this spot does not show prominently after ripening begins because of the natural dark color of the prune. A striking characteristic is the exudation of a drop of gum from each spot or pit. This exudation of gum resembles the effect produced by insect punctures in the skin of the fruit, but in this case no insect or other parasite is responsible. In addition to these external symptoms, and sometimes unaccompanied by any outward evidences of disease, there appear groups of dead brown cells scattered irregularly through the flesh of the fruit. These spots bear a strong resemblance to cork in apples. In more severe cases the surface of the fruit becomes shriveled and corrugated, with brown discoloration extending into the flesh. (Reference 1.)

**Spray injury.** — Spraying is universally recommended for the control of a great many plant diseases, but the use of sprays is not without its

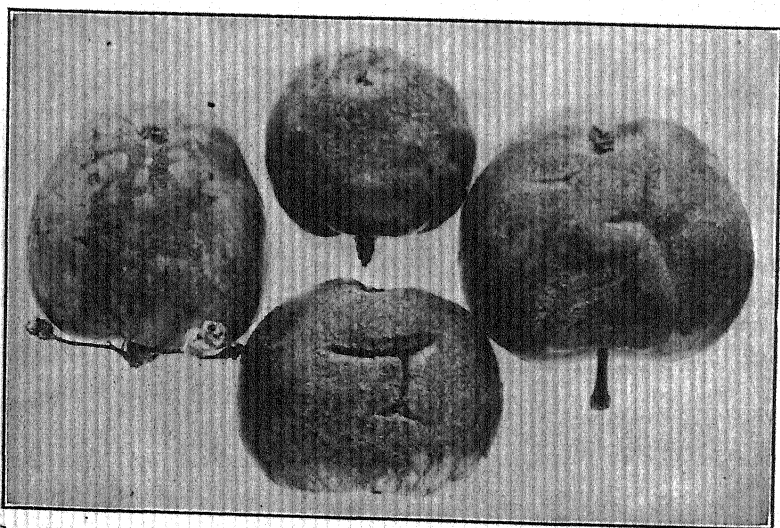


FIG. 221. — An extreme case of bordeaux spray injury on apple fruits. (Photograph by Jackson, Ore. Agr. Exp. Sta.)

drawbacks in the way of injury resulting to the sprayed plants. The difficulty in the use of sprays is to obtain a preparation that will kill the parasite and not injure the host. Bordeaux, lime-sulfur, the arsenical compounds and the oil sprays have all given more or less trouble at times and under certain conditions. Bordeaux is apt to cause leaf

injury and russetting of fruit (Fig. 221) in moist weather, and lime-sulfur sometimes causes burning of fruit and foliage in hot weather. Severe burning of foliage sometimes results from some of the arsenical sprays. (References 8, 12, 25, 26.)

**Freezing injury.** — There are many types of injury to plants resulting from low temperatures. Some of these forms are indicated by such terms as winter-injury, freezing-injury, and frost-injury. Damage resulting from freezing ranges all the way from that caused by untimely late spring frosts to blossoms, new shoots and young fruits, to the killing of trees by severe freezes in winter and the losses sustained when fruits and vegetables are allowed to freeze in storage. Fruit crop failures are common because of late spring frosts which kill the blossoms. Frosts occurring after the fruit is set may result in more or less serious russet injury to the fruits if they are not killed outright. Winter injury to trees ranges from the killing of twigs to the formation of winter "sunscald" cankers and the actual bursting of the bark due to sudden hard freezing when the tree is full of sap, or even the outright killing of the tree. (References 2, 3, 9, 13, 16, 17, 20.)

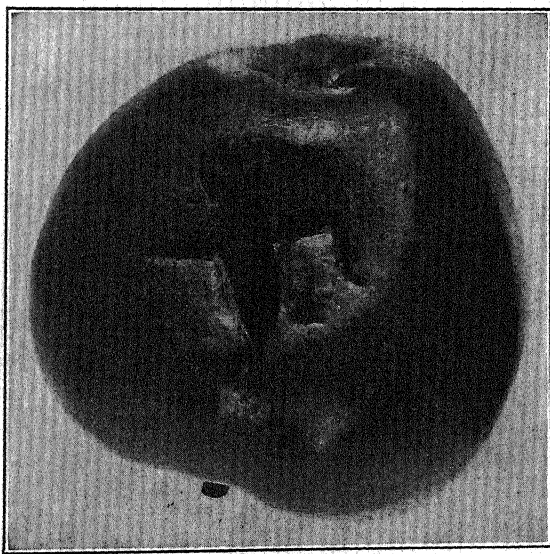


FIG. 222. — Sunburn on apple. The burning occurred in June. The subsequent growth of the fruit caused deep cracks to appear in the burned area.

**Sunburn.** — Sometimes an extremely hot spell of weather in midsummer results in the actual burning of fruits. Sunburned spots occur on the cheek of the fruit that is exposed to the most direct rays of the sun.

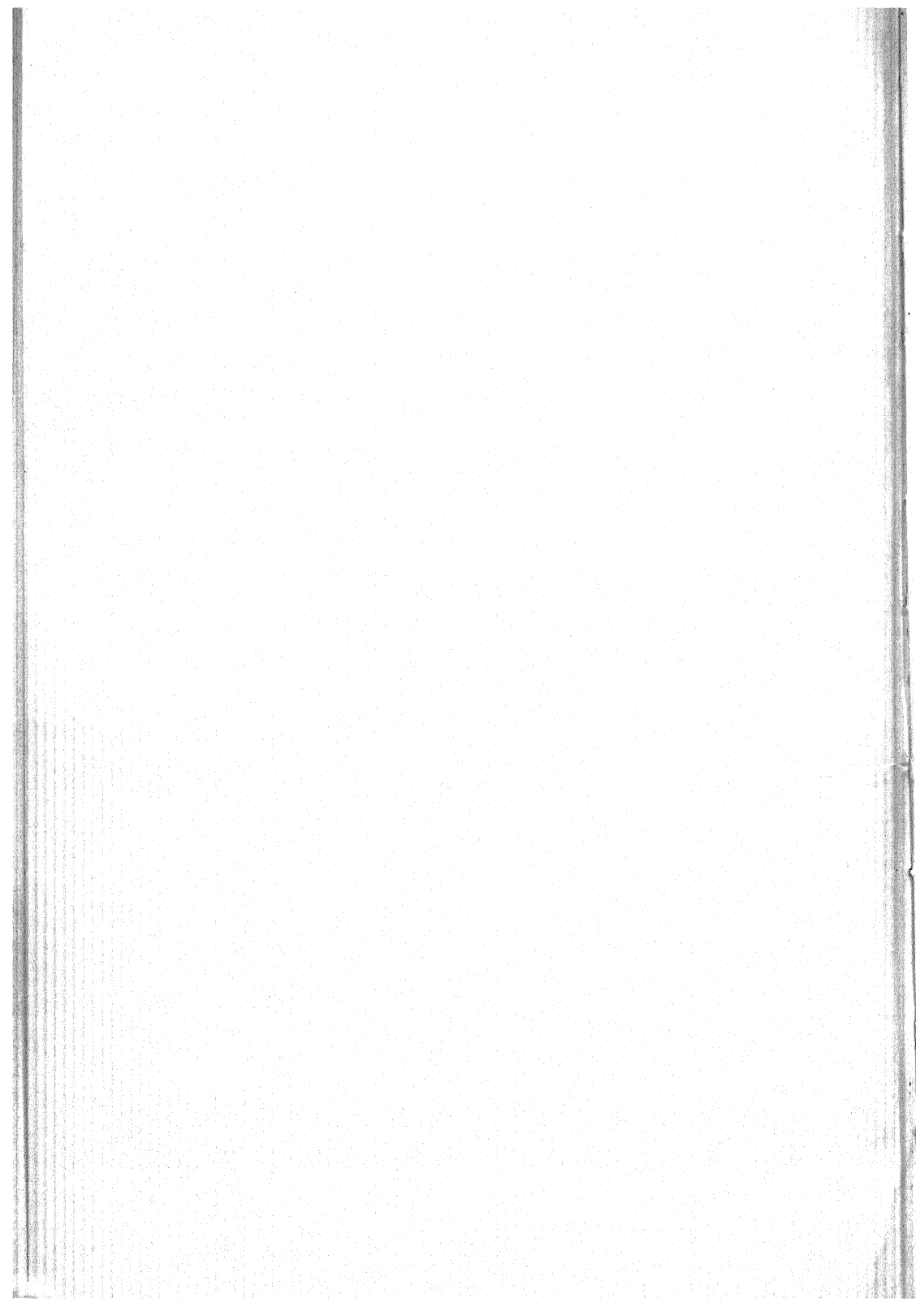
Different kinds of fruits are subject to sunburn. Figure 222 illustrates a badly sunburned apple. This burning occurred in June before the fruit was nearly mature. The flesh was actually cooked to a depth of a few millimeters. Further growth of the apple causes the burned surface to crack. Tomatoes, melons and cucumbers are frequently seen with sunburned spots on the surface most directly exposed to the sun's rays.

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